



**HAZARDOUS  
SITE CONTROL  
DIVISION**

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Planning/  
Field  
Investigation  
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(REM/FIT)**

**ZONE II**

**CONTRACT NO.  
68-01-6692**

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June 19, 1986

W66204.DO

Mr. Larry Rexroat, RPM  
Environmental Protection Agency  
Region VI  
InterFirst Two Building  
1201 Elm Street  
Dallas, Texas 75270

Dear Larry:

We are pleased to submit thirty (30) copies of the Final Offsite Feasibility Study for the Vertac site. Additional copies are distributed as indicated below.

The report has been revised to address your oral comments of June 3.

Sincerely,

Richard G. Saterdal, P.E.  
Site Project Manager

DE/VERTC7/038/nkm

Enclosures (30)

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## EXECUTIVE SUMMARY

The objective of this endangerment assessment is to evaluate the potential health and environmental effects if no remedial action is taken at the offsite area adjacent to the Vertac Chemical Corporation, Jacksonville, Arkansas. The offsite area includes the sewage collection and treatment system, abandoned and west facilities; Rocky Branch, Bayou Meto, and flood plains of Rocky Branch and Bayou Meto. The assessment defines the current or potential future problems attributable to contaminants at the site and primarily 2,3,7,8-tetrachlorodibenzodioxin (TCDD).

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This endangerment assessment includes a discussion of the available data and how it is used. Soil, sediment, and fish were sampled and analyzed for TCDD, and in some cases chlorophenoxy herbicides, chlorinated benzenes, and chlorinated phenols. Historical data for the site was also considered to identify contamination trends. Concentrations of compounds identified in soils and sediments were compared to background concentrations of compounds to determine if the contaminant concentrations in the investigation area exceeded expected or normal concentrations for the area.

A discussion of the potential for migration of TCDD from the sewer system, Rocky Branch, and Bayou Meto is included. It concludes that TCDD has the potential to migrate out of the sewage treatment plant, will adsorb onto soils and sediments and can be transported in the creek beds and flood plains.

Potential exposure pathways to contaminated media include: direct dermal contact or ingestion of sediments or soils originating from the sewer system, Rocky Branch, Bayou Meto, or the flood plains of Rocky Branch and Bayou Meto; inhalation of volatilized organics from contaminants in the sewer

system, creek, or flood plain sediments or soils; ingestion of fish and other aquatic organisms from Rocky Branch or Bayou Meto; and ingestion of agricultural products that have been grown in contaminated soils.

From the estimation of intakes and considering various exposure scenarios, risks were quantified. The scenario of residential use of the flood plain presents the highest estimated risks for ingestion of TCDD contaminated soils. Risks ranged from  $10^{-3}$  to  $10^{-7}$  for the various scenarios.

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Section 1  
INTRODUCTION

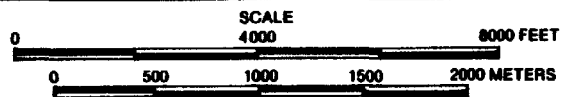
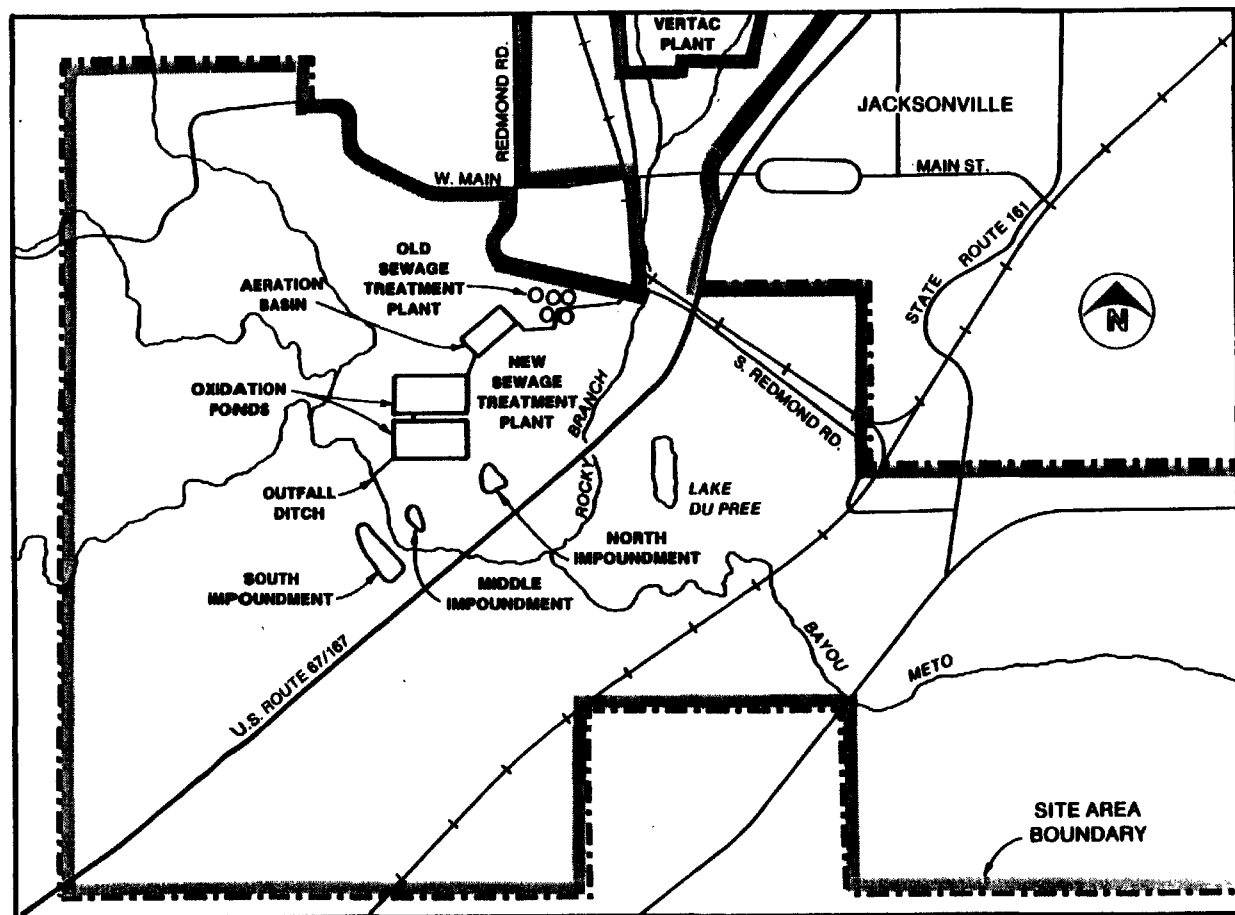
The objective of this endangerment assessment is to evaluate the potential health and environmental effects if no remedial action is taken at the offsite area adjacent to the Vertac Chemical Corporation, Jacksonville, Arkansas. The assessment will define the current or potential future problems attributable to contaminants at the site. This evaluation of the no-action alternative is performed in compliance with the Comprehensive Environment Response, Compensation and Liability Act of 1980 (CERCLA), Section 104, which requires protection of public health and the environment.

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For a risk to exist, each of the following components must exist:

- o A source of contamination
- o Release of the contaminant to a transport media
- o Transport of the contamination to a receptor
- o Exposure of the receptor to the contaminant
- o An adverse effect resulting from the exposure

The following sections will determine if each of these components are present and evaluate the consequences of no remedial activities at the sewage collection and treatment system, either the old or the new; Rocky Branch, Bayou Meto, and flood plains of Rocky Branch and Bayou Meto. The area referred to as offsite in this evaluation is shown on Figure 1-1.



008572 **FIGURE 1-1**  
**INVESTIGATION AREA MAP**

The results of the extent of contamination in the offsite area are presented in Chapter 5 and discussed in Chapter 6 of the Final Offsite Remedial Investigation report (RI).

The prime contaminant of concern identified onsite was 2,3,7,8-tetrachlorodibenzo-p-dioxin, or TCDD. The purpose of the offsite remedial investigation was to determine the extent of TCDD migration away from the site. The RI was limited in scope. The only media samples were soils, sediments and aquatic biota. Media were preferentially analyzed for TCDD, although limited analyses for contaminants other than TCDD were conducted. Special studies, all relating to determining the amount of soils and sediments potentially contaminated, were conducted. Data from these studies are useful in delineating the physical features, important for determining media interaction and transport.

This endangerment assessment begins with a discussion of the available data and how it is used for the purposes of site evaluation. Average background concentrations are presented for comparison to concentrations found in the study area. Applicable or relevant and appropriate requirements are compared to concentrations found onsite.

A summary of the potential for migration of contaminants is made, taking into account; the physical features of the site, the physical and chemical characteristics of the contaminants, the fate and transport of the contaminants in the environment and potential pathways for migration.

The exposure assessment then defines exposure pathways specific to conditions of the study area and receptor populations. A toxicity assessment identifies toxicological properties of the contaminants present. The exposure assessment,

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toxicity assessment and previous sections are then integrated in an assessment of risk.

Risk from exposure depends on the magnitude of the exposure, the duration of exposure and the chemicals' toxicity. Quantitative estimates of risk are made where ever data are available. Qualitative assessments of risk are discussed for pathways or contaminants where quantitative estimates cannot be made.

The limitations, assumptions, and uncertainties inherent in the development of risk estimates are defined. Methodologies used in this assessment are presented in Appendix A.

A summary of the findings of the potential endangerment of public health or the environment from the offsite areas adjacent to the Vertac Chemical Corporation conclude the assessment.

Appendix C contains a report by the Agency for Toxic Substances and Disease Registry (ATSDR) of the Centers for Disease Control (CDC) on the "Health Assessment, Offsite Remedial Investigation, Vertac Chemical Corporation." This report recommends future sampling studies, evaluates the current data, and recommends action levels for site remediation.

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Section 2  
DATA EVALUATION AND LIMITATIONS

Data used in this assessment are those presented in Tables 4-8 and 5-1 in the Final Offsite Remedial Investigation (RI) report and consist of:

- o 74 soil and sediment samples, analyzed for TCDD, chlorophenoxy herbicides, toluene, chlorinated benzenes, and chlorinated phenols were taken along the water courses, in water bodies in sewer lines and in the sewage treatment plant (old and new) in December 1983. TCDD was detected in 40 of the 74 samples. The detection limit varied between 0.005 and 0.015 ppb.
- o 21 soil and sediment samples analyzed for TCDD only were taken south of Bayou Meto in June 1984. Only 1 sample had measurable concentrations of TCDD. Detection limits were not given in Table 5-1 of the RI.
- o 258 soil and sediment samples were taken throughout the offsite area in August 1984. Of the 258, 29 were field blanks, 225 were analyzed for TCDD (4 were replicates), 54 were analyzed for 2,4-D and 2,4,5-T, and 55 were analyzed for volatile organics, base/neutrals and PCB/pesticides. Out of the 225 analyzed for TCDD, 79 had measurable concentrations and 150 were reported as at the detection limit for TCDD (ranging between 0.01 and 3.41 ppb) or the concentration was estimated. Of the 74 samples taken in the flood plain, 13 had measurable concentrations of TCDD and 61 had concentrations below

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the detection limits of 0.02 to 0.9 ppb, but were estimated. Thirty-two of the flood plain samples were taken south of Bayou Meto. Only 1 of these samples had a measurable concentration of TCDD.

- o 4 background samples and blanks were taken 3 miles north and upgradient of the investigation area during the December 1983 sampling. All samples were less than the quantification limit which averaged 0.011 ppb.
- o 27 fish samples taken from Bayou Meto in August 1984. These were analyzed as either whole fish samples or fillets. Whole fish concentrations were higher than fillets. Method detection limits ranged from 2.4 to 18 ppt.

Historical data on TCDD from the period June 1975 through May 1983 exists for the offsite area. These data are useful for analysis of contamination trends, but will not be used in this assessment of potential endangerment. Advances in analytical methodologies and decreasing detection limits make compatibility of historical data with present data questionable.

In addition, the creeks and flood plains of the investigation area are a very dynamic ecosystem. Continual flooding causes resuspension and deposition of the sediments, and dilution of contamination. The location and concentration of contaminants a year ago may not be representative of what is presently occurring at the site. Therefore, only the latest data, August 1984, will be used in determining the risk from exposure to contaminants in the creeks and flood plains.

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A trend analysis was conducted as part of the RI and is discussed in Chapter 6 of the RI. No pattern of TCDD contamination was apparent in the flood plains. This analysis supports the decision to use only the latest data in the endangerment assessment. The trend analysis for TCDD contamination in the sewers indicated a continual increase over the years in locations where repeated sampling had occurred. Therefore, the August 1984 sampling is the most current in the trend and will be used in subsequent sections. The sewers represent a continued source of contaminants and it is not known if the concentrations reported in 1984 are indicative of the highest concentrations in the system.

The data collected during the Vertac offsite RI are subject to a number of limitations. Because the suspected areas of contamination were so vast, previously collected data were evaluated and used to limit the scope and cost of the RI. Based on these data, the RI was focused on the most toxic and persistent contaminant, TCDD. Only a very cursory investigation was made for other contaminants.

While TCDD data for the sewer lines and sewage treatment plant were consistently above quantifiable limits, most of the TCDD data from the waterways and flood plains were at or below quantifiable limits and some were not accepted by the Quality Assurance Check. These data did not provide a sufficient data base for determining the precise levels and extents of contamination in the waterways and flood plains, but did indicate contamination was present.

Limited analysis for contaminants other than TCDD was conducted during the RI. The Quality Assurance/Quality Control procedures conducted during the RI, concluded the data was not valid and should not be used. This assessment will consider these compounds qualitatively, noting their presence

but not quantities. This analysis supports the decision to use only the latest data in the endangerment assessment.

Data are grouped by area and media; sewer system, Rocky Branch, Bayou Meto, and flood plains, and soils/sediments or fish. Average and maximum reported concentrations of TCDD from the August 1984 sampling in the specific media are presented in Tables 2-1 through 2-3. Average values are calculated using detection limits, quantification limits, and estimated values when the data are available. Use of other than measured concentrations tends to over estimate average concentrations. Maximum reported concentrations indicated whether the value was a measured concentration, at the detection limit, quantification limit or an estimated value. Tables 2-1 and 2-2 indicate the total number of measured concentrations observed at each sampling location during the 1984 period.

The detection limit is the minimum concentration of a substance that can be identified, measured, and reported, with 99% confidence that the analyte concentration is greater than zero. The detection limit is determined from analysis of a sample in a given matrix containing the target analyte. The detection limit will vary as a function of sample type and size. Detection limits are indicated by a "d" next to the concentration.

The estimated maximum concentration is used when accurate quantification of the analyte cannot be accomplished due to interferences in the sample. The result reported is calculated assuming that the total instrument response is due to the analyte. The actual analyte concentration is probably less than that reported. Estimated concentrations are indicated by an "e" next to the concentration.

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Table 2-1  
AVERAGE AND MAXIMUM REPORTED TCDD CONCENTRATIONS  
IN THE WASTEWATER COLLECTION AND TREATMENT SYSTEMS,  
AUGUST 1984

Location	TCDD Concentration (µg/kg)				Total No. of Samples Taken	Total No. of Measured Concentration
	0-3 Inches		3-6 Inches			
	Max.	Ave.	Max.	Ave.		
Collection System	200 <sup>a</sup>	28.1	1.7 <sup>b</sup>	-- <sup>e</sup>	18	12
West STP Aeration Basin	37.9	20.2	--	--	4	4
West STP Oxidation Pond, North	3.6	2.8	--	--	6	3
West STP Oxidation Pond, South	1.3	1.2	--	--	8	2
West STP Outfall Ditch	0.19 <sup>d</sup>	0.13	--	--	4	0
Abandoned STP Digester	NS	NS	12.46	8.88	2	2
Abandoned STP Clarifiers	1.62	0.93	--	--	2	2
Abandoned STP Drying Beds	0.77	0.39	6.59	3.59	4	3
Abandoned STP Sludge Collection Area	0.08 <sup>c</sup>	0.06	--	--	6	2

<sup>a</sup> actual value greater than 200.00

<sup>b</sup> only value detected, no average possible

<sup>c</sup> detection limits

<sup>d</sup> estimated value

<sup>e</sup> no 3-6 inch sample taken

NS = not sampled

\* STP = Sewage Treatment Plant

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Table 2-2  
AVERAGE AND MAXIMUM REPORTED TCDD CONCENTRATIONS IN ROCKY  
BRANCH, BAYOU METO, AND FLOOD PLAINS, AUGUST 1984

Location	TCDD Concentration (µg/kg)						Total No. of Samples Taken	Total No. of Measured Concentration
	0-3 Inches		3-6 Inches		6-9 Inches			
	Max.	Ave. <sup>a</sup>	Max.	Ave. <sup>a</sup>	Max.	Ave. <sup>a</sup>		
Rocky Branch instream	0.74 <sup>d</sup>	0.23	0.39 <sup>b</sup>	0.13	0.75 <sup>c</sup>	0.18	25	10 <sup>0</sup>
Rocky Branch near stream	0.84 <sup>d</sup>	0.56	0.13 <sup>b</sup>	--	7.58	1.46	15	3 <sup>1</sup>
Bayou Meto instream	0.79 <sup>c</sup>	0.38	0.21 <sup>b</sup>	--	0.33 <sup>c</sup>	0.195	25	12 <sup>0</sup>
Bayou Meto near stream	3.5 <sup>d</sup>	1.44	1.52	0.81	2.1	0.73	24	13 <sup>0</sup>
2-yr flood plain, north of Bayou Meto	1.7	0.51	0.24 <sup>b</sup>	--	1.58	0.34	27	10 <sup>0</sup>
5-yr flood plain, north of Bayou Meto	0.9 <sup>c</sup>	0.16	NS	NS	0.83 <sup>c</sup>	0.25	15	0
2-yr flood plain, south of Bayou Meto	0.9	0.21	NS	NS	7.79 <sup>c</sup>	0.85	22	1
5-yr flood plain, south of Bayou Meto	0.06 <sup>c</sup>	0.03	NS	NS	0.06 <sup>c</sup>	0.05	10	0

<sup>a</sup> Average calculated with detection limits and estimated concentrations.

<sup>b</sup> only value detected, no average available

<sup>c</sup> detection limit

<sup>d</sup> estimated value

NS = not sampled

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Table 2-3  
MAXIMUM AND AVERAGE REPORTED CONCENTRATIONS OF TCDD IN FISH  
SAMPLES AT VARIOUS LOCATIONS, AUGUST 1984

Location	Total No. of fish	Sample Desc.	TCDD Concentration (ug/g)	
			Max.	Ave.
BM-02-upstream of R.B. confluence	9	whole	176	97
RB-01-Bayou Mato confluence with R.B.	3	whole	798	319
BM-03-2.5 miles downstream of confluence with R.B.	2	whole	863	711
BM-03-2.5 miles downstream of confluence with R.B.	3	fillet-no skin	136 <sup>a</sup>	--
BM-040-Bayou Mato at Highway 31 (13 miles downstream of Rocky Branch)	2	whole abs. fillets	157	83
BM-04-Bayou Mato @ Highway 31	1	fillet - no skin	<5.7 <sup>ab</sup>	--
BM--04-Bayou Mato @ Highway 31	1	fillet - with skin	30 <sup>a</sup>	--
BM-05-Bayou Mato at Highway 79 (250 miles downstream of Rocky Branch)	3	whole	7 <sup>ab</sup>	--
BM-05-Bayou Mato at Highway 79	3	fillet-no skin	9 <sup>d</sup>	6
BM-05-Bayou Mato at Highway 79	1	whole abs. fillet	11 <sup>a</sup>	--
BM-06-Bayou Mato at Highway 152 (275 miles downstream of Rocky Branch)	3	whole abs. fillets	36	20
BM-06-Bayou Mato at Highway 152	1	fillets-with skin	10	10
BM-06-Bayou Mato at Highway 152	3	fillets-no skin	5 <sup>a</sup>	--
BM-07-Bayou Mato at Wildlife Man. Area	1	whole abs fillets	28 <sup>a</sup>	--
BM-07-Bayou Mato at Wildlife Man Area	1	fillets-with skin	9	9
BM-07-Bayou Mato at Wildlife Man Area	1	whole	19 <sup>a</sup>	--

<sup>a</sup>Only one sample, therefore average not calculated.

<sup>b</sup>Detection limit.

Note: Total number of fish does not indicate total number of samples at that location.  
One sample could be composed of more than one fish.

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The quantification limit is arbitrarily defined at some level above the detection limit. It is affected by sample size and dilution. Quantification limits are indicated by a "q" next to the concentration.

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Section 3  
STANDARDS COMPARISON

Standards and criteria do not exist for compounds found in soils and sediments. Concentrations of compounds found in soils and sediments can be compared to background concentrations of compounds in an effort to determine if the investigation area exceeds what "normally" occurs in the area. Four background samples were taken 3 miles north and upgradient of the investigation area. All background samples yielded concentrations of TCDD at the quantification limits. The average of these samples is 0.01 ppb. This value can be compared against average and maximum concentrations of TCDD from Tables 2-1 and 2-2.

Various agencies have made recommendations on "acceptable" levels of TCDD in soils and fish tissue. These recommendations have been made on specific sites under a specific set of assumptions that may or may not apply to other sites.

The Centers for Disease Control (CDC) has recommended that TCDD concentrations not exceed 1 ppb in residential soils (U.S. EPA, 1984). CDC's recommendation was made for a residential setting, where continual contact with soils would occur over a 70-year lifetime from infancy to old-age. They have also recommended this level for residential soils and channel sediments and flood plain soils which are subject to erosion and transport processes in this study area (Appendix C). CDC also recommends that TCDD concentrations not exceed 5-7 ppb in soils and sediments where the general public may have infrequent contact (Appendix C).

The Food and Drug Administration (FDA) has recommended that concentrations of TCDD less than 25 ppt in fish pose no serious health threat and intake of these amounts not be

restricted (U.S. EPA, 1984). Canada has established a limit of 20 ppt of TCDD in fish exported to the United States. The New York State Department of Health has unofficially proposed a 10 ppt limit of TCDD in fish as a ceiling for human consumption (U.S. EPA, 1984). These recommendations may or may not be applicable to the site. The methodology used in the calculation of acceptable limits in fish tissue is based upon concentration in the water body, type of fish species, fish consumption rate for the area where fish are caught and other assumptions specific to the receptors. These may or may not apply to conditions at Bayou Meto.

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### STANDARDS ANALYSIS

#### Sewer System

Maximum and average concentrations of TCDD detected in the sewer system at various locations are presented in Table 2-1. Maximum and average concentrations detected in all locations in the system were greater than background concentrations of the general area. The maximum concentration detected was greater than 200 ppb.

#### Rocky Branch

Less than half (40 percent) of the instream samples and 20 percent of the near stream samples had measurable concentrations of TCDD.

Maximum and average concentrations of TCDD found instream and near stream in the 0 to 6 inch depth are above background concentrations.

#### Bayou Meto

Approximately half (48 percent) of the instream samples and slightly over half (54 percent) of the near stream samples

had measurable concentrations of TCDD.

Maximum and average concentrations of the instream samples at all depths taken were above background. All near stream concentrations exceed background.

Both average and maximum reported concentrations of TCDD in fish exceed the FDA recommendation of 25 ppt in fish samples taken at the confluence of Rocky Branch and Bayou Meto and for 2.5 miles downstream of the confluence. Concentrations in fish upstream of the confluence also exceed this recommendation.

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Concentrations of TCDD in fish samples at Bayou Meto and Highway 79 and further downstream were generally lower than the 25 ppt recommendation, except in two maximum reported concentrations of whole fish samples. All average concentrations from this point were lower than 25 ppt.

#### Flood Plain

Within the 2-year flood plain, north of Bayou Meto, 37 percent of the samples taken had measurable concentrations of TCDD. The average maximum reported concentration in the 0- to 3-inch and 6- to 9-inch depths exceed background. Within the 5-year flood plain, north of Bayou Meto, none of the 15 samples had measurable concentrations of TCDD. All were reported at the detection limits on the samples. All detection limits were greater than background detection limits.

Within the 2-year flood plain south of Bayou Meto, 5 percent of the samples taken had measurable concentrations of TCDD. Both maximum and average concentrations within the 0- to 3-inch depth were above background concentrations. No samples

were taken in the 3- to 6-inch depth. No measurable concentrations were detected in the 6- to 9-inch depth; however, the maximum detection limit was high, 7.79 ppb. Within the 5-year flood plain south of Bayou Meto, no measurable concentrations of TCDD were detected. All of the concentrations were at the detection limits however all detection limits were greater than background.

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## Section 4

### CONTAMINANT MIGRATION

The objective of this section is to evaluate the potential for migration of TCDD from the sewer system, Rocky Branch and Bayou Meto. Factors influencing potential migration include: the physical characteristics of the sewers; the physical characteristics and hydrologic setting of Rocky Branch and Bayou Meto; the physical properties of TCDD; and the physical, chemical and biological transformation process by which TCDD is transported and/or transformed in the environment.

#### PHYSICAL FACTORS AFFECTING SEDIMENT MIGRATION

The degree to which sediments can be transported in any medium is dependant on several factors. Stream flow is a major factor with high flows scouring stream sediments and carrying (entraining) them downstream. The physical and chemical characteristics of the sediment and entraining water directly influence the type and volume of material eroded and transported and the condition of deposition when and where it occurs. Both entrainment and transport depend on the shape, size, and weight of the particle and the forces exerted on the particle by the flow of water. When these forces are diminished to the extent that the transport rate is reduced or transport is no longer possible, deposition occurs.

Sediment is transported in suspension (wash load) or as bed load, which rolls or slides along the bed of the creek. Sediment moves interchangeably by suspension and bed load. The rate of bed load transport depends on the availability of sediment and the influence of change in water flow conditions. If the supply of sediment is reduced by erosion control the amount of bed material or sediments will be

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reduced, especially during high flow conditions. Suspended sediment load includes both the bed material load in suspension and the wash load. Where the chief source of sediment is fine-textured soils, the wash load rather than the total bed material load usually constitutes the majority of the sediment discharge. Lower water velocities are required to keep the finer sediments in suspension and moving downstream. The coarser material requires higher water velocities before it is entrained or moves as bed load. Coarse material will not be moved as rapidly as the finer material.

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#### PHYSICAL FEATURES OF THE SEWER SYSTEM

The first sewage treatment plant, constructed about 1941 was a gravity fed, sanitary sewer system. The same system is in operation today with the exception of the old treatment plant and 4,600 feet of the Rocky Branch Interceptor from manhole (MH) 20 through MH 3. In 1961 the treatment plant was upgraded by rehabilitating the pump station and clarifiers, and adding a sludge digester, sludge drying beds, and 44 acres of oxidation ponds. Solids removal from the aeration or oxidation ponds does not occur. The Rocky Branch Interceptor was replaced in 1979 because of infiltration problems. The new line runs parallel to and immediately east of the old line. The sewer collection system is shown on Figure 3-12 in the Final RI report.

In 1961 the sewage treatment plant began accepting wastes from the Vertac site. An outfall line was constructed to convey wastes from the site to the Rocky Branch Interceptor. However, many of the sewer lines at the Vertac site were constructed in 1941 and likely conveyed wastes from the site prior to the 1961 agreement. It is possible that some of the old lines continued to convey wastes after 1961 agreement and the construction of the outfall line to Rocky Branch

Interceptor. For example, a single line, from MH 80, may still transfer sewage from the Vertac Plant. This line passes through the residential subdivision immediately south of the Vertac site before reaching the treatment plant.

The sewer lines from the Vertac Plant are all clay pipe, from 6 inches to 12 inches in diameter. Six of the 14 manholes on the plant site have structural defects, ranging from initial construction defects, missing bricks, to broken or missing manhole covers. These defects may lead to infiltration, exfiltration and sediment accumulation. Infiltration is a problem at MH 126 because of missing bricks below the rim, and flow into the manhole from a portion of Rocky Branch diverted from the cooling ponds on site. Deterioration of MH 120 may be responsible for infiltration problems. For more specific data on problems in the sewer system refer to Figure 4-14 and Table 4-6 of the Final RI report.

Lines in the residential area south of the Vertac Plant site consist of 6-inch concrete laterals joining to 10-inch to 16-inch clay interceptors. Most of the subdivision lines are off-line and off-grade. Some obstructions occur in these lines due to grease, vines, and fallen bricks and concrete from deterioration of manholes. Laterals at lower elevations suffer from infiltration and blockage. Overflow of MH 106, west of the subdivision and 200 feet south of the Vertac Plant site has been observed (Draft RI 1985).

The new Rocky Branch Interceptor line is constructed of 15-inch to 24-inch clay pipe. No data are available to determine if connections exist between the old line and the new line. The old line is currently filled with sediment and water. It is plugged on the north end at MH 20, but the plug is missing on the south end at MH 3. Surcharging may occur at MH 7 as it runs at capacity. Surcharging is typically caused by insufficient hydraulic capacity; pipe

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constriction either from root intrusion, sediment deposition or collapsed pipe; structural problems; and/or excessive infiltration.

Infiltration into sewer lines can occur if groundwater is at the levels of the lines or excessive rainfall has occurred. Lines are usually built with a drainage channel for directing water away from the pipe but it is not known if this is the case in this area. Exfiltration of water and sediments through defective joints and cracks in the pipe from the sewer lines to groundwater may also occur.

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#### PHYSICAL FEATURES AND HYDROLOGIC SETTING

##### Rocky Branch

Rocky Branch flows south from the northern boundary of Jacksonville, traversing the Vertac Plant property along the west side and empties into Bayou Meto approximately 2 miles south of the Plant. Minor intermittent streams flow into Rocky Branch during periods of heavy rainfall in the spring.

The stream is characterized by low sinuosity, low levels of suspended sediments, and a high bed-load potential. Sediment load of Rocky Branch is derived from erosion of upgradient and surrounding terrain. Average sediment depth is about 10 inches. Channel deposits are predominantly silt and clay. Generally, both banks are steep, but there are occasional small point bars at meanders. Lag gravels are found on point bars and along the upper reaches of the stream. As the stream approaches Bayou Meto, the channel becomes wider and sediments become finer.

Flooding occurs frequently on Rocky Branch, which causes sediment deposition along the irregular flood plain. A

flood plain delineation study was performed as part of the RI. Section 4.1 of the RI details methodology and results of the study. Delineation of the 2-year and 5-year flood plain is shown on Figure 7-1 of the Final RI report. Flood elevations and discharge for various locations along Rocky Branch are shown on Table 4-1.

#### Bayou Meto

Bayou Meto begins approximately one mile northwest of Jacksonville. At the Fall Line, a northeast-southwest-trending boundary of major physiographic and geologic provinces, the stream changes course from south to east and becomes broad and sinuous. As the gradient decreases, the water flow turns sluggish. Abandoned and partly filled channels with inter-connecting oxbow lakes, ponds, and minor tributaries are common along the entire reach of Bayou Meto.

Sediments are generally fine grained sand, silts, and clays. Gravel deposits are uncommon, primarily because of the low flow conditions. Organic composition is high because of vegetation decay. Bayou Meto joins the Arkansas River approximately 130 miles southeast of Jacksonville.

Flooding occurs frequently along Bayou Meto. Sediment deposition is common along the extensive irregular flood plains. Delineation of the 2-year and 5-year flood plain is shown on Figure 7-1 of the Final RI report. The flood plain is bisected by U.S. Highway 67/67, State Highway 161, and the Missouri-Pacific railroad line, all of which are raised on earthen artificial embankments. Flood elevations and discharge for various locations along Bayou Meto are shown on Table 4-1.

Table 4-1  
FLOOD ELEVATIONS AND DISCHARGE AT SPECIFIC LOCATION ON ROCKY  
BRANCH AND BAYOU METO

<u>Location</u>	<u>Discharge (cfs)</u>		<u>Elevation (ft)</u>	
	<u>2-year</u>	<u>5-year</u>	<u>2-year</u>	<u>5-year</u>
Rocky Branch downstream of Vertac	349	629	256.0	256.0
Rocky Branch intersection with Redmond Road	521	940	243.4	244.2
Bayou Meto influence on Rocky Branch (0.76 miles upstream of confluence with B.M.)	521	940	243.0	243.7
Confluence at R.B.	521	940	243.3	244.0
Confluence at B.M.	2,610	10,500	241.9	246.5
Intersection of B.M. with Highway 16	2,550	9,300	238.3	242.0

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## PROPERTIES OF THE CONTAMINANT

### Physical Features

Physical and chemical characteristics of TCDD are presented on Table 4-2. Appendix B presents a discussion of the chemical properties of all contaminants.

### Fate and Transport

Several definitions will be useful in the discussions below:

- o Solubility. The solubility of a chemical is a measure of its tendency to dissolve in water. Many physical properties influence the solubility of a chemical in the environment: valence, state, temperature, soil adsorption, and presence of other chemicals.
- o Vapor pressure. Vapor pressure represents the tendency of a chemical to volatilize from the pure substance. Although this ideal condition is not likely to be present in the environment, the parameter provides an indication of the importance of volatilization.
- o Octanol-water partition coefficient. This parameter expresses the tendency of a chemical to concentrate in either the organic or aqueous phase. The coefficient correlates with the bioconcentration factor.
- o Soil-water partition coefficient. This parameter is the equilibrium ratio of the chemical concentration in soil to the concentration in water divided by the soil organic carbon and provides an

Table 4-2  
PHYSICAL/CHEMICAL CHARACTERISTICS OF CONTAMINANT

<u>Contaminant</u>	<u>Molecular Weight</u>	<u>Solubility in mg/L</u>	<u>Vapor Pressure mm Hg @ 25°C</u>	<u>Octanol/Water Par. Coef.</u>	<u>Soil/Water Par. Coef.</u>	<u>Biocon. Factor</u>
2,3,7,8-TCDD	321.9	0.0002	0.000001	6.9E+06 - 1.9E+07	3.8E+06	2.0E+03 to 3.0E+04

<sup>a</sup> at 53.78°F

<sup>b</sup> value is log K<sub>ow</sub>

Source: ICF. 1985.

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indication of the leachability of a chemical in the soil.

- o Bioconcentration factor. This parameter is the equilibrium ratio of the chemical concentration in fish to the concentration in water at equilibrium. The coefficient represents the tendency of a chemical to concentrate in the tissue of aquatic species.

TCDD is a relatively stable compound in the environment, exhibiting relatively strong resistance to biodegradation. The half-life of TCDD in soils due to biodegradation has been estimated to be 10-12 years (U.S. EPA 1985). Photodegradation in the presence of UV light has been shown to occur in air and dry soils. It is not considered to be a predominant fate in aquatic environments. TCDD has very low water solubilities and a high octanol/water partition coefficient and soil/water partition coefficient, indicating its tendency to sorb to sediments and biota in an aquatic media. It does not readily evaporate from an aquatic medium, partly because of its strong affinity for sediments and partly because of its low vapor pressure. In soils, TCDD tends to remain near the surface and over time, TCDD sorbed to soils becomes more difficult to desorb.

As TCDD is likely to be present only in the sorbed state in the environment, it would therefore, be transported primarily with soils, such as in dust entrainment, erosion into surface water, and suspension and resedimentation in aquatic media. Based on available data, the potential for vertical movement of TCDD in soils to the groundwater is negligible under most conditions.

TCDD will bioaccumulate in aquatic organisms, with experimental bioconcentration factors ranging from 2,000 to 30,000.

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It has also been demonstrated that once the source of TCDD is removed, the half-life of TCDD in fish flesh is approximately one year (Ecology UŠA, 1985).

#### CONTAMINANT MIGRATION POTENTIAL

From the above discussions, it is apparent that TCDD has the potential to migrate out of the sewage treatment plant, will adsorb to soils and sediments and can be transported in the creek beds and flood plains. Various potential pathways of migration, considering the physical site conditions and contaminant properties include:

- o     Runoff and releases from the Vertac site discharging via surface runoff to the Rocky Branch
- o     Overflows of the sewer collection system, discharging to surface soils
- o     Backup of interceptor lines into residences
- o     Exfiltration from defective sewer lines (i.e., joints, cracks, manholes) to groundwater
- o     Interconnections between the abandoned old interceptor line coming from the Vertac Plant and the new line
- o     Sewage Treatment Plant Lagoon and oxidation pond overflows
- o     Discharge to Rocky Branch from the old sewage treatment plant via an old conduit

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- o Discharge to Bayou Meto from the new sewage treatment plant via the outfall
- o Flooding along Bayou Meto and Rocky Branch causing resuspension and deposition of sediments
- o Flooding causing inundation and overflow of the lagoons and oxidation ponds
- o Wind entrainment of flood plain soils

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Section 5  
EXPOSURE ASSESSMENT

This section provides a brief toxicity assessment of TCDD and then examines the environmental pathways by which exposure could occur. Exposures to all types of contaminants that may be present are assessed. Potential receptors are identified. Exposure settings are developed for the sewer system, creeks and flood plains. Each setting is discussed in terms of the events that must occur for human exposure and the potential duration of exposure.

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TOXICITY ASSESSMENT

A summary of the toxicological properties of TCDD is presented below. For a detailed discussion of the toxicology of all the contaminants identified in the RI refer to Appendix B.

2,3,7,8-Tetrachlorodibenzo-p-dioxin.

TCDD has been shown to be extremely toxic in experimental animals and has teratogenic, mutagenic and carcinogenic effects. The International Agency for Research on Cancer (IARC) has classified TCDD as a positive animal carcinogen and probable human carcinogen but insufficient data exists on human carcinogenicity (2B). U.S. EPA Carcinogen Assessment Group (CAG) has classified it as a probable human carcinogen with a combination of sufficient evidence in animals and inadequate data in humans (Group B2).

Sensitivity and target organs differ for various species. Chloracne and hyperkeratosis are distinctive symptoms of TCDD exposure in animals and humans. A number of reports

suggest an association of soft tissue sarcomas and TCDD exposure in humans.

Toxicological profiles above are based on exposure to a single compound, and synergistic and antagonistic effects between many compounds have not been considered. The state of toxicological knowledge is insufficient to describe the potential effects of multiple exposure to a variety of contaminants. This assessment assumes that the effects of multiple compound exposure are additive.

#### EXPOSURE PATHWAYS

Potential exposure pathways to contaminated media include:

- o Direct dermal contact or ingestion of sediments or soils originating from the sewer system
- o Inhalation of volatilized organics from contaminants in the sewer system
- o Direct dermal contact with or ingestion of sediments from Rocky Branch or Bayou Meto
- o Direct dermal contact with or ingestion of soils of the flood plain of Rocky Branch and Bayou Meto
- o Inhalation of volatilized organics from creek or flood plain sediments and soils
- o Ingestion of fish and other aquatic organisms from Rocky Branch or Bayou Meto that have bioaccumulated contaminants

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contaminants in soils or vegetations are accumulated in animal tissue.

### RECEPTORS

The investigation area is located within and adjacent to the City of Jacksonville, Arkansas. Jacksonville has a total population of 27,589 people according to the 1980 census. There are 7,378 families and 8,787 households.

The population in the area of investigation outside Jacksonville is estimated to be about 3,000. The area is a mixture of residential and agricultural with extensive undeveloped, uninhabited woodland and swamp land. The potential exists for any of these persons to come in contact with contaminants.

In identifying potentially exposed populations, it is desirable also to identify sensitive groups such as persons who may be occupationally exposed to TCDD, women of childbearing age, and pregnant women. These groups have been selected because they are particularly sensitive because of the toxic effects of TCDD on reproductive and developing systems (fetotoxic and teratogenic). The distribution of these groups within the general population of Jacksonville or those within the area of investigation is not known.

Employees who operate and maintain the wastewater collection and treatment systems could potentially be exposed to contaminants in the sewer system in addition to those in the creeks and flood plains.

Persons who consume dairy products from cattle pastured in the flood plains, produce grown in the flood plains or in the sludge-drying beds, or fish from the bayou have the potential for exposure to contaminants. No estimates on the number of persons potentially exposed are available.

Other potential receptors include those persons utilizing water from Bayou Meto downstream of the area of investigation for irrigation and livestock. Table 4-5 in the RI lists surface water withdrawal points for all uses in the Jacksonville area. The number of persons potentially affected is not known.

No municipal water is drawn from surface water in this area.

The definition of no action includes the potential for future development. Future residential development of most of the area is not likely as the major portion of the investigation area is within the 2- to 5-year flood plain. Industrial development is also unlikely. Continued agricultural usage is probable.

#### ESTIMATES OF INTAKE

Estimates of media intake on a daily basis or lifetime average daily intake are fairly standardized and accepted. For example, a 2 L/day water ingestion rate for a 70 kg adult and 6.5 g/day ingestion rate for freshwater aquatic organisms are recognized by U.S. EPA as acceptable values to use (ICF, 1985). The lifetime average daily intake is an age and body weight adjusted estimate that takes into account the different ingestion rates and body weights for the different age groups.

While estimates for water and fish are standardized, estimates for soil intake by adults and children vary widely. Daily soil ingestion rate estimates for children range from as high as 10 g/day (Kimbrough et al., 1984) downward to 0.10 g/day

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(Lepow et al., 1974). Recent work by the CDC suggests a soil ingestion rate for 1 to 3 year olds ranging from 0.18 to 1.8 g/day (Binder et al., 1985). Daily soil ingestion rate estimates for adults range from 1 g/day (Kimbrough et al., 1984) to zero (Schaum, 1984). Appendix A shows the derivation of these estimates.

The maximum reported concentrations of contaminants in each medium are used in estimation of intake rates. While this may result in an overestimation of risk, the use of maximum reported concentrations represents a conservative approach and is an attempt to assess the highest individual potential exposures. Arithmetic means are also used to estimate a more realistic exposure.

#### EXPOSURE SETTINGS

##### Sewage Treatment System

Potential exposure to contaminants in the sewer system could occur through: surcharging of sewer lines into residential areas just south of the Vertac Plant Site, contact with sediments through the use of the sludge drying beds as garden plots, treatment plant worker contact with sediments during maintenance activities, and continued migration of sediments in the sewer system to Bayou Meto through the sewer line outfall.

Contaminants in the sewers pose a potential threat to the environment through continued migration of contaminated sediments into the creeks and flood plains. Concentration of all contaminants were greatest in the sewer system than any other area in the investigation area. In turn, a human health hazard is posed by the continued migration of contaminants to areas where human contact is a possibility. The pathway

of continued migration from the sewers to the creeks and flood plains cannot be quantified. The RI has estimated the total potentially contaminated sediment accumulated in the sewage collection system to be approximately 47 cubic yards, 500 cubic yards in the old treatment plant facilities and an additional 214,000 cubic yards in the aeration basin and oxidation ponds. It is not possible to quantify how much sediment would be transported to the creeks and flood plains, or at what concentrations.

An occupational setting will be used to quantify risk from exposure to sediments in the sewer system. It is assumed that workers will come into contact with sediments somewhere in the system each day during cleaning and maintenance activities.

An assumption of the setting is that exposure to sediment is reduced to zero when temperatures are below freezing or precipitation is greater than 0.01 inch as frozen or wet sediments do not become wind-borne, and gloves are worn when temperatures are cold.

Workers can be exposed to contaminants in the sediments through ingestion of sediments through dust entrainment or contact with hands, and subsequently into the mouth; inhalation of volatile organics; inhalation of dusts; and dermal absorption through contact with sediments.

Adult worker daily soil intake rates have not been documented. In this assessment, a daily soil intake of 0.1 g/day from dust ingestion and soil accumulation on hands is used. This makes the lifetime daily average intake of soil for workers 0.0008 g/kg body weight/day.

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Intake rates cannot be quantified for dermal absorption. Intake rates will be not quantified for inhalation of volatiles or dust because air quality data are lacking.

### Rocky Branch

The primary exposure routes to sediments in stream and near stream at Rocky Branch are ingestion and dermal contact from recreational use of the creek and exposure to residents whose yards are adjacent to the creek. The residential exposure setting is discussed in the following section on "Flood Plains." The exposures that would occur in the recreational setting, would be intermittent and would differ over a lifetime. For example, a person may play at the site as a child, swim and fish as an adolescent, and hunt and fish as an adult. Each activity would result in different exposure due to differences in amount of contact with soil and sediment and hygiene habits. Sediments ingested are assumed to result from accumulation on the hands. Dermal contact results from hand and foot contact with sediments.

Childhood exposure is based on an intake of 0.1 g/day over the ages 5 to 18 years out of a 70-year lifetime, soil exposure two days per week, 6 months per year, and an average body weight of 38 kg. Adult exposure is based on an intake of 0.1 g/day over the ages 18 to 60 years out of a 70-year lifetime, soil exposure two days per week, 6 months per year, and an average body weight of 70 kg. A combined exposure of both the child and adult is also estimated. Lifetime average soil intake for the above settings is 0.00007 g/kg-day, 0.00012 g/kg-day and 0.00019 g/kg-day, respectively.

Intake rates for dermal exposures are not quantified in this assessment. Determination of surface area exposed during various activities at different life stages, quantity, and duration of contact leads to a high degree of uncertainty.

### Bayou Meto

The exposure setting and intake estimates are the same for Bayou Meto as outlined above for Rocky Branch.

Bayou Meto has an additional exposure route, that of fish ingestion. This setting assumes that all fish consumed are from the bayou and fish are eaten on a daily basis equal to that of the U.S. average for freshwater fish, 6.5 grams per day. However, surveys have shown that individual total fish consumption varies as much as threefold. In this assessment, a daily fish intake of 6.5 g/day is used and the lifetime daily average intake for the adult years, 18 to 60 years only, is 0.06 g/kg body weight/day.

For purposes of this evaluation, it is assumed that the source of contamination to the Bayou is not controlled; therefore, fish will remain contaminated over the exposure period, assumed to be a 70-year lifetime.

### Flood Plains

Potential exposure to contaminants in the flood plain soils could occur through residential or recreational use of the area. Potential routes are through ingestion, inhalation and dermal contact with soils. Residents living on or adjacent to the flood plains (including along Rocky Branch) could ingest contaminated soils during outdoor activities or soils may be transported into homes by wind, on pets, or on hands and clothing. Ingestion is assumed to occur through soil contact with the hands and subsequent hand-to-mouth contact. Lifetime soil ingestion rates were estimated to be about 0.028 g/kg body weight/day based on a variable body weight and ingestion rate over a 70-year lifetime. Appendix A describes the methodology used in the calculation of the

lifetime ingestion rate. Estimates of intake through dermal exposure were not made for this assessment.

A recreational setting assumes exposures are intermittent, limited by climatological factors and activities over a lifetime. Childhood exposure is based on an intake of 0.1 g/day over the ages 5 to 18 years, soil exposure 2 days per week, 6 months per year, an average body weight of 38 kg and a 70-year lifetime. Adult exposure is based on an intake of 0.1 g/day over the ages 18 to 60 years, soil exposure 2 days per week, 6 months per year, an average body weight of 70 kg and a 70-year lifetime. A combined exposure of both the child and adult is also estimated. Lifetime average soil intake for the above settings is 0.00007 g/kg-day, 0.00012 g/kg-day and 0.00019 g/kg-day, respectively.

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Section 6  
RISK ASSESSMENT

INTRODUCTION

TCDD is classified as a B2 carcinogen by the Carcinogen Assessment Group (CAG) of the U.S. EPA. This classification indicates it is a probable human carcinogen because of sufficient evidence in animals, although inadequate evidence in humans.

Carcinogens

If a compound is considered a carcinogen by CAG, a cancer potency is derived for a specific exposure route. Potency values are not available for all carcinogens due to the lack of adequate data. A cancer potency is derived from the upper 95th percent confidence limit on the response per unit intake of a compound over a lifetime (i.e., only 5 percent chance that the probability of response could be greater than the estimated value for the experimental data used). TCDD has a potency value of  $1.56 \times 10^5$  (mg/kg/day)<sup>-1</sup> for ingestion.

This potency value is used in conjunction with the lifetime average daily intake of a compound (via the same exposure route used to derive the potency value) in the calculation of excess cancer risk. Excess lifetime cancer risk is defined as the incremental increase in the probability of developing cancer compared to the background probability (if no exposure occurred). Carcinogenic risk is calculated by the equation:

$$r = 1 - e^{-pd}$$

Where:

r = risk

p = potency

d = dose

e = base of the natural system of logarithms (2.71828)

This equation can be used where chemical intakes may be large or small because it does not assume risk is directly related to intake or that the dose-response relationship is linear. For example, a  $1 \times 10^{-6}$  excess lifetime cancer risk would represent the risk resulting from an exposure that is associated with an increase in cancer incidence by one case per million people exposed.

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## LIMITATIONS AND ASSUMPTIONS

### Limitations

The risk assessment process involves a good deal of uncertainty. Uncertainty is a function of the completeness of the site data, judgements used in determining exposure settings, accuracy of the toxicity data base, appropriateness of the cancer risk models used to develop potency factors, and assumptions used to simplify site conditions in order to approximate exposure concentrations. A list of general and specific uncertainty factors is presented in Table 6-1. When dealing with uncertainties in assessing risk to the public, it is reasonable to use a conservative approach, which will lead to an expected overestimate of the actual risk.

### Assumptions

Public health assessments utilize various assumptions, generalizations and standardized values in assessing risk.

Table 6-1  
GENERAL UNCERTAINTY FACTORS IN RISK ASSESSMENT

Uncertainty Factor	Effect of Risk Estimation		
	Over- estimate	Under- estimate	Over/under estimate
Exposures through dust or vapor inhalation and dermal absorption are not quantified.		X	
The exposure to and concentration of contaminants at exposure points is held constant over a 70-year lifetime. Chemical fate and transport mechanisms may alter actual concentrations which may vary with time.			X
Risks are assumed to be additive. Risks may not be additive because of synergistic or antagonistic actions of other chemicals.			X
Cancer potencies are primarily derived using laboratory animal studies and, when available, human occupational studies. Extrapolation of data from high to low dose, from one species to another, and from one exposure route to another all use conservative estimates.			X
Cancer potencies are based on upper 95th percentile slope. It represents the upper bound of the model used.	X		
The exposure to soil is based on assumptions regarding climatological conditions (such as precipitation and days of frozen soil).			X

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Table 6-1  
(continued)

Uncertainty Factor	Effect of Risk Estimation		
	Over-estimate	Under-estimate	Over/under estimate
Chemical concentrations reported as "detected but less than the quantification limit" in the RI report are used in this endan-germent assessment as equal to the quantification limit.	X		
All intake of TCDD is assumed to come from media being evaluated. Does not take into account other sources (i.e., no relative source contribution).		X	
Not all chemicals found at the site are quantified. Therefore, risk cannot be calculated. They could contribute to an adverse health risk.		X	
The assumptions regarding body weight, average lifetime exposed, intake of contaminants, population characteristics, and lifestyle may not be representative for any actually exposed population.			X
This assessment is based on the present understanding of the site characteristics from 1984 data. Conditions at the site or understanding of the site may change over time.			X

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These are listed in Table 6-2 along with assumptions specific to the exposure settings.

## RISK CHARACTERIZATION

### Sewer System

The occupational setting assumes workers are exposed to sediments found in the sewer system during cleaning and maintenance activities. Exposure would be limited and may in reality be zero. An exposure fraction is calculated on the basis of climatic conditions that would inhibit dust generation and temperatures that would require gloves to limit hand-to-mouth contact. (See Appendix A). An absorption fraction of 0.3 through the gastrointestinal tract is used in determining risk. Risk from contact with sediment that is likely to result in ingestion (using the maximum reported concentrations of TCDD) is estimated to be  $3 \times 10^{-3}$ . Risk from ingestion using of the average concentrations at various locations in the sewage treatment system ranges from  $10^{-4}$  to  $10^{-7}$ . Specific values are presented in Table 6-3.

Risks from dermal contact with TCDD in sediments and inhalation of TCDD laden dusts are not quantitatively assessed. Data are lacking to determine dermal absorption rates, exposure areas and potency values for dermal exposures. Inhalation absorption rates, potency values and dust concentrations are also unavailable. Exposure to TCDD by these routes would add to the excess cancer risk posed by sediments in the sewer system.

Exposure to contaminants other than TCDD that are present but not quantifiable, may increase the risk of adverse health effects.

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Table 6-2  
RISK ASSESSMENT ASSUMPTIONS

Assumption	Comment
The site has the potential for unrestricted future recreational use and fishing.	Part of the definition of no action.
Exposure to contaminants remains constant over lifetime.	Conservative assumption - Life style changes and actual residence time would alter exposures.
Concentration of contaminants remains constant over the exposure period.	Conservative assumption. Environmental fate and transport can change concentrations.
Years in lifetime = 70 Working years = 40 Years of Recreational Use Child = 13 Adult = 42 Residential years = 70 Adult body weight = 70 kg Child body weight = 10 kg	Standard values used by U.S. EPA in deriving risk. (U.S. EPA 1980 and ICF 1985.)
Soil Intake 0.1 gram/day = adult 0.1 gram/day = average child (5-18)	Kimbrough et al. (1984) Kimbrough et al. (1984)
Fish Intake 6.5 gram/day = adult	ICF (1985)
Lifetime average daily intake (LADI): Soil (LASI) = 0.0008 for occupational to 0.00007 g/kg body weight/day Soil (LASI) = 0.0002 to 0.00007 recreational Fish (LAFI) = 0.06 k/kg body weight/day	This is an age and time weighted rates over a 70-year lifetime to account for the relatively higher ingestion rates per kg of body weight in younger age classes (see Appendix A).
Media intake represents 100% of contaminant intake.	There may be other occupational or environmental sources of the compound.
Fish ingestion only occurs during adult years and all fish consumed are caught from Bayou Meto.	All fish consumed may not be from the Bayou. This may act to overestimate the risk.

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Table 6-2  
(continued)

Assumption	Comment
Climatic conditions reduce outdoor activities and dust generation and hence, exposure.	See Appendix A.
Values of less than quantification limit are assumed to be equal to the quantification limit.	Conservative assumption to assess upper bound risk (i.e, not to underestimate risk).

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Table 6-3  
EXCESS LIFETIME CANCER RISK FROM INGESTION OF SEDIMENTS  
FROM THE SEWAGE SYSTEM, OCCUPATIONAL SETTING

Location	Excess Cancer Risk @ TCDD Concentration (mg/kg) <sup>a</sup>			
	0-3 inches		3-6 inches	
	Risk at Reported Maximum	Risk At Average	Risk at Reported Maximum	Risk at Average
Collection System	3E-03	4E-04	2E-05	
New STP Aeration Basin	6E-04	3E-04		
New STP Oxidation Pond				
North	5E-05	4E-05		
New STP Oxidation Pond				
South	2E-05	2E-05		
New STP Outfall Ditch	3E-06	2E-06		
Old STP Digest			2E-04	1E-04
OLD STP Clarifiers	2E-05		1E-05	
Old STP Drying Beds	1E-05	6E-06	1E-04	5E-05
Old STP Sludge				
Collection Area	1E-06	9E-07		

<sup>a</sup> Concentrations found on Table 2-1.

Notes: LASI = 0.0008 g/kg body weight/day  
                     (Appendix A)  
 Cancer Potency = 156000 (mg/kg/day)<sub>1</sub>  
                     (EPA 1985 )  
 Exposure Fraction = 0.39 (Appendix A presents  
                     derivation)  
 Absorption Fraction = 0.3 (Schaum 1984)

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## Rocky Branch

Exposures to sediments in the Rocky Branch area could occur in both the recreational and residential settings. Existing residences along the Rocky Branch may be subject to flood events. Some of the residential yards incorporate the Rocky Branch creek banks as part of the yard and lack physical barriers between the yard and the creek. Sections along the Rocky Branch are also used for recreation.

A recreational setting assumes exposure to in-stream and near stream sediments. Ingestion is assumed to occur through sediment contact with the hands and subsequent contact with the mouth. The absorption fraction in the gut is estimated at 0.3.

Exposure is limited due to climatic conditions which reduce the amount of time spent out of doors. Risk from ingestion of sediment at the maximum reported concentration of TCDD found in sediments at any depth in the childhood setting is  $1 \times 10^{-5}$ . Using maximum concentration found at any depth and the adult setting, the risk is estimated at  $3 \times 10^{-5}$ . Using maximum concentration found at any depth and the combined setting, the risk is estimated at  $4 \times 10^{-5}$ . Risk from ingestion using the average concentrations in each setting is presented in Tables 6-4, 6-5, and 6-6.

In the resident setting, risk from ingestion of sediment at the maximum reported concentration of TCDD found in sediments at any depth is  $6 \times 10^{-3}$ . Risks from ingestion of sediment using average concentrations in specific locations on Rocky Branch are presented in Table 6-7.

Risks from dermal contact with TCDD in sediments in either the recreational or residential settings are not quantitatively assessed. Data are lacking to determine dermal

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Table 6-4  
EXCESS CANCER RISK DUE TO TCDD INGESTED IN SOIL/SEDIMENTS  
DURING CHILDHOOD YEARS AT VARIOUS LOCATIONS

Location	Excess Cancer Risk at Various TCDD Concentrations (ug/kg) and Locations <sup>a</sup>					
	0-3 Inches		3-6 Inches		6-9 Inches	
	Risk at Reported Maximum	Risk at Average	Risk at Reported Maximum	Risk at Average	Risk at Reported Maximum	Risk at Average
Rocky Branch instream	1E-06	4E-07	7E-07	3E-07	1E-06	3E-07
Rocky Branch near stream	2E-06	1E-06	2E-07		1E-05	3E-06
Bayou Meto instream	2E-06	7E-07	4E-07		6E-07	4E-07
Bayou Meto near stream	7E-06	3E-06	3E-06	2E-06	4E-06	1E-06
2-yr Floodplain, North of Bayou Meto	3E-06	1E-06	5E-07		3E-06	7E-07
5-yr Floodplain, North of Bayou Meto	2E-06	3E-07			2E-06	5E-07
2-yr Floodplain, South of Bayou Meto	8E-07	4E-07			2E-05	2E-06
5-yr Floodplain, South of Bayou Meto	1E-07	6E-08			1E-07	1E-07

<sup>a</sup> Concentrations found on Table 2-2

Notes: Lifetime average soil ingestion = 0.00007 g/kg body weight/day. (See Appendix A)  
Cancer potency = 156000 (mg/kg/day)<sup>-1</sup> (EPA 1985)  
Exposure fraction = 0.58 (Appendix A)  
Absorption fraction = 0.3 (Schaum 1984)

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Table 6-5  
EXCESS CANCER RISK DUE TO TCDD INGESTED IN SOIL/SEDIMENT  
DURING ADULT YEARS AT VARIOUS LOCATIONS

<u>Excess Cancer Risk at Various TCDD Concentrations (ug/kg) and Locations<sup>a</sup></u>						
<u>Location</u>	<u>0-3 Inches</u>		<u>3-6 Inches</u>		<u>6-9 Inches</u>	
	<u>Risk at Reported Maximum</u>	<u>Risk at Average</u>	<u>Risk at Reported Maximum</u>	<u>Risk at Average</u>	<u>Risk at Reported Maximum</u>	<u>Risk at Average</u>
Rocky Branch instream	2E-06	8E-07	1E-06	4E-07	2E-06	6E-07
Rocky Branch near stream	3E-06	2E-06	4E-07		2E-05	5E-06
Bayou Mato instream	3E-06	1E-06	7E-07		1E-06	6E-07
Bayou Mato near stream	1E-05	5E-06	5E-06	3E-06	7E-06	2E-06
2-Year Floodplain, North of Bayou Mato	6E-06	2E-06	8E-07		5E-06	1E-06
5-Year Floodplain, North of Bayou Mato	3E-06	5E-07			3E-06	8E-07
2-Year Floodplain, South of Bayou Mato	1E-06	7E-07			3E-05	3E-06
5-Year Floodplain, South of Bayou Mato	2E-07	1E-07			2E-07	2E-07

<sup>a</sup> Concentrations found on Table 2-2

Notes: Lifetime average soil ingestion = 0.00012 g/kg body weight/day. (See Appendix A)  
 Cancer potency = 156000 (mg/kg/day)<sup>-1</sup> (EPA 1985f)  
 Exposure fraction = 0.58 (Appendix A)  
 GSC Absorption fraction = 0.3 (Schaum 1984)

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Table 6-6  
EXCESS CANCER RISK DUE TO TCDD INGESTED IN SOIL/SEDIMENT  
DURING CHILDHOOD AND ADULT YEARS AT VARIOUS LOCATIONS

Location	Excess Cancer Risk at Various TCDD Concentrations (mg/kg) and Locations <sup>a</sup>					
	0-3 Inches		3-6 Inches		6-9 Inches	
	Risk at Reported Maximum	Risk at Average	Risk at Reported Maximum	Risk at Average	Risk at Reported Maximum	Risk at Average
Rocky Branch instream	4E-06	1E-06	2E-06	7E-07	4E-06	9E-07
Rocky Branch near stream	4E-06	3E-06	7E-07		4E-05	8E-06
Bayou Meto instream	4E-06	2E-06	1E-06		2E-06	1E-06
Bayou Meto near stream	2E-05	7E-06	8E-06	4E-06	1E-05	4E-06
2-Year Floodplain, North of Bayou Meto	9E-06	3E-06	1E-06		8E-06	2E-06
5-Year Floodplain, North of Bayou Meto	5E-06	8E-07			4E-06	1E-06
2-Year Floodplain, South of Bayou Meto	2E-06	1E-06			4E-05	4E-06
5-Year Floodplain, South of Bayou Meto	3E-07	2E-07			3E-07	3E-07

<sup>a</sup> Concentrations found on Table 2-2

Notes: Lifetime average soil ingestion = 0.00019 g/kg body weight/day. (See Appendix A)  
 Cancer potency = 156000 (mg/kg/day)<sup>-1</sup> (EPA 1985f)  
 Exposure fraction = 0.58 (Appendix A)  
 GSC Absorption fraction = 0.3 (Schaum 1984)

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Table 6-7  
EXCESS CANCER RISK DUE TO TCDD INGESTED FROM FLOODPLAIN  
SOILS AT VARIOUS LOCATIONS AND DEPTHS  
IN A RESIDENTIAL SETTING

Location	Excess Cancer Risk at Various TCDD Concentrations (ug/kg) and Locations <sup>a</sup>					
	0-3 Inches		3-6 Inches		6-9 Inches	
	Risk at Reported	Risk at Average	Risk at Reported	Risk at Average	Risk at Reported	Risk at Average
	Maximum	Average	Maximum	Average	Maximum	Average
Rocky Branch Instream	6E-04	2E--04	3E-04	1E-04	6E-04	1E-04
Rocky Branch Near Stream	6E-04	4E-03	1E-04		6E-03	1E-03
2-Year Floodplain, North of Bayou Mato	1E-03 (3E-05)	4E-04 (9E06)	2E-04 (4E-06)		1E-03 (3E-05)	2E-04 (5E-06)
5-Year Floodplain, North of Bayou Mato	7E-04 (2E-05)	1E-04 (3E-06)			6E-04 (2E-05)	2E-04 (5E-06)
2-Year Floodplain, South of Bayou Mato	7E-04 (2E-05)	2E-04 (4E-06)			6E-03 (8E-05)	6E-04 (2E-05)
5-Year Floodplain, South of Bayou Mato	4E-05 (1E-06)	2E-05 (5E-07)			4E-05 (9E-07)	4E-05 (1E-06)

<sup>a</sup> TCDD Concentrations found on Table 2-2

Notes: Lifetime average soil ingestion = 0.028 g/kg body weight/day. (See Appendix A)  
 Cancer potency = 156000 (mg/kg/day)<sup>-1</sup> (EPA 1985f)  
 Exposure fraction = 0.58 (Appendix A)  
 GSC Absorption fraction = 0.3 (Schaum 1984)

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absorption rates, exposure areas and potency values for dermal exposures. Exposure to TCDD by this route would add to the excess cancer risk posed by sediments in Rocky Branch.

Exposure to contaminants other than TCDD that are present but not quantified may increase the risk of adverse health effects.

### Sediments

A recreational setting assumes exposure to in-stream and near stream sediments. Ingestion is assumed to occur through sediment contact with the hands and subsequent contact with the mouth. The absorption fraction in the gut has been estimated to be 0.3.

Exposure is limited due to climatic conditions which reduce the amount of time spent out of doors. Risk from ingestion of sediment at the maximum reported concentration of TCDD found at any depth in the childhood setting is  $7 \times 10^{-6}$ . Using maximum reported concentration found at any depth and the adult setting, the risk is estimated at  $1 \times 10^{-5}$ . Using the maximum concentration found at any depth and the combined setting, the risk is estimated at  $2 \times 10^{-5}$ . Risk from ingestion using of the average concentrations in each setting is presented in Tables 6-4, 6-5, and 6-6.

Risks from dermal contact with TCDD in sediments are not quantitatively assessed. Data are lacking to determine dermal absorption rates, exposure areas and potency values for dermal exposures. Exposure to TCDD by this route would add to the excess cancer risk posed by sediments in the bayou.

Exposure to contaminants other than TCDD that are present but not quantified may increase the risk of adverse health effects.

### Fish Ingestion

Considering the 0.3 absorption fraction in the gut, risk from ingestion of fish with the maximum reported concentrations of TCDD found at any point in the bayou is  $7 \times 10^{-3}$ . Risks from ingestion of fish using average concentrations in specific locations on the Bayou are presented in Table 6-8 and range from  $10^{-3}$  to  $10^{-5}$ .

### Flood plains

Using a residential setting, risk from ingestion of soils in the flood plain north of Bayou Meto at the maximum reported concentration of TCDD found at any depth is  $1 \times 10^{-3}$ . Ingestion of soils south of the bayou could result in an excess cancer risk of  $6 \times 10^{-3}$ . Climatological factors limit exposures and have been included in the estimation of risk. Risk from ingestion of soils using average concentrations, both north and south of the bayou is presented in Table 6-8.

Using a recreational setting risk from ingestion of soils north of Bayou Meto at the maximum reported concentration of TCDD found at any depth in the childhood setting is  $3 \times 10^{-6}$ . Using the maximum reported concentration found at any depth and the adult setting, the risk is estimated at  $6 \times 10^{-6}$ . Using the maximum reported concentration found at any depth and the combined setting, the risk is estimated at  $9 \times 10^{-6}$ .

Risk from ingestion of soils south of Bayou Meto at the maximum reported concentrations of TCDD found at any depth in the childhood setting is  $2 \times 10^{-5}$ . Using the maximum reported concentration found at any depth and the adult setting, the risk is estimated at  $3 \times 10^{-5}$ . Using the maximum reported concentration found at any depth and the combined setting, the risk is estimated at  $4 \times 10^{-5}$ .

Table 6-8  
EXCESS CANCER RISK DUE TO TCDD INGESTED IN FISH  
DURING THE ADULT YEARS FROM BAYOU METO

Location	Excess Cancer Risk	
	Risk at Max. Conc. (ppt)	Risk at Ave. Conc. (ppt)
BM-02 - Upstream of R.B. confluence	1E-03	8E-04
RB-01 - Bayou Meto confluence w/R.B	6E-03	3E-03
BM-03 - 2.5 miles downstream of confluence w/R.B.	7E-03	6E-03
BM-03 - 2.5 miles downstream of confluence w/R.B.	1E-03	
BM-05 - Bayou Meto at HWY 79		6E-05
BM-05 - Bayou Meto at HWY 79	7E-05	5E-05
BM-05 - Bayou Meto at HWY 79	9E-05	
BM-06 - Bayou Meto at HWY 152	3E-04	2E-04
BM-06 - Bayou Meto at HWY 152	8E-05	8E-05
BM-06 - Bayou Meto at HWY 152	4E-05	
BM-07 - Bayou Meto at Wildlife Man. Area	8E-05	7E-05
BM-07 - Bayou Meto at Wildlife Man. Area	1E-04	
BM-07 - Bayou Meto at Wildlife Man. Area	2E-04	

Notes: Lifetime average fish intake = 0.056 g/kg body  
weight/day (Appendix A)  
Cancer potency = 156000 (mg/kg  
body weight/day)<sup>-1</sup> (EPA 1985f)  
GSC Absorption fraction = 0.9 (Schaum 1984)

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Risk from ingestion of soils using the average concentrations, both north and south of the Bayou, in each setting is presented in Tables 6-4, 6-5, and 6-6.

Risks from dermal contact with TCDD in sediments and inhalation of TCDD laden dusts are not quantitatively assessed. Data are lacking to determine dermal absorption rates, exposure areas and potency values for dermal exposures. Inhalation absorption rates, potency values, and dust concentrations are also unavailable. Dust emissions are likely to be a localized problem because the area is heavily vegetated. Exposure to TCDD by these routes may add to the excess cancer risk posed by soils in the flood plain.

Exposure to contaminants other than TCDD that are present but not quantifiable may increase the risk of adverse health effects.

#### SUMMARY

A summary of the risks presented by each route is shown in Table 6-9. Using the assumptions given, the scenario of residential use of the flood plain and Rocky Branch present the highest estimated risks for ingestion of TCDD contaminated soils. This scenario assumes children between the ages of 1.5 and 3.5 consume 10 grams of soil per day, 1g/day from 3.5 to 5, and adults consume 0.1 grams per day. Other investigators believe children consume no more than 0.1 grams of soil per day and adults do not consume any soil (Lepow 1974; Schaum 1984). If this were the case, risk from the residential scenario would drop by one and a half orders of magnitude.

For sediments found in the sewer system, if daily contact was to occur and ingestion of 0.1 grams of soil per day resulted risks could be as high as  $10^{-3}$  in areas of maximum

Table 6-9  
SUMMARY OF SITE PROBLEMS AND ASSOCIATED RISK

Contaminated Media	Pathway	Assessment
Sewer System Sediments	Direct/ Ingestion	Risk ranges from $10^{-3}$ to $10^{-6}$ using occupational settings. Contact with sediments in the system on a daily basis is unlikely.
	Dermal	Was not quantified, may act to increase total risk. This is the most likely pathway for worker exposure to sediments within the sewer system.
	Inhalation	Was not quantified, may act to increase total risk. Inhalation of volatiles is a possibility. Quantification of volatiles was not done in the RI.
	Indirect/ Ingestion, Dermal, Inhalation	Was not quantified. Could occur through overflow, backflow, exfiltration, etc. However, it is anticipated to be a minor risk
	Migrating to creeks	Was not quantified. Anticipated to present a substantial risk to environment.
Rocky Branch Sediments	Direct/ Ingestion	Risks range from $10^{-3}$ to $10^{-4}$ using the residential scenario and Kimbrough estimates of childhood soil intake. Risk ranges from $10^{-6}$ to $10^{-7}$ using the recreational scenario, 0-3" sediment depth and any age group.
	Dermal	
	Indirect/ Secondary Contact (pets, etc.)	Pathway was not quantified. May act to increase the total risk. Pathways not quantified. Limited risk anticipated
	Aquatic Uptake	Pathway not quantified. Data not available to determine risk to aquatic life.
Bayou Meto Sediments	Direct/ Ingestion	Risk ranges from $10^{-5}$ to $10^{-7}$ using the recreational scenario, 0-3" sediment depth and any age group. Risk is about the same for all sediment depths.

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Table 6-9  
(continued)

Contaminated Media	Pathway	Assessment
	Dermal	Pathway was not quantified. May act to increase the total risk.
Bayou Meto (cont'd.)	Indirect/ Secondary Contact (pets, etc.)	Pathways not quantified. Limited risk anticipated.
Fish	Direct/ Ingestion	Risk ranges from $10^{-3}$ to $10^{-4}$ using the adult consumption setting. Risk is lower using TCDD concentrations in fish below 2.5 miles downstream of the confluence with Rocky Branch.
	Dermal	Pathway not quantified. Limited risk anticipated.
Floodplains	Direct/ Ingestion	Risk ranges from $10^{-3}$ to $10^{-5}$ using the residential scenario and Kimbrough estimates of childhood soil intake. Risk ranges from $10^{-6}$ to $10^{-8}$ using the recreational scenario, 0-3" sediment depth and any age group. Risk is slightly higher for the 6-9" soil depth due to one maximum concentration ( $10^{+5}$ ).
	Dermal	Pathway was not quantified. May act to increase the total risk.
	Inhalation	Pathway was not quantified, anticipated to be minor increase in total risk. Dust entrainment of soils in the floodplain not anticipated to be high due to dense vegetative cover.
	Indirect/ Leaching to Ground- water	Not quantified. Considered not a major risk due to mobility of TCDD. No data available to assess pathway.

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concentration. It is unlikely that daily contact with sediments would occur, even during cleaning and maintenance activities. Risks presented in this scenario represent a conservative "worst-case" approach to estimating actual health risks.

Sediments in the in-stream and near-stream areas of Rocky Branch and Bayou Meto present a minor public health risk from direct contact with contaminated sediments under the recreational setting using the given assumptions. Risk may be present to aquatic organisms from contaminated sediment, but this pathway is not quantifiable. The interaction between TCDD in sediments and water is not known. Some solubilization may be possible over time. Aquatic organisms may bioconcentrate TCDD up to 30,000 times the surrounding water concentration. Bottom feeders may ingest TCDD contaminated sediments directly during feeding. Data are insufficient to determine the effects of TCDD on aquatic organisms as fish have been found with over 850 ppt in their tissues with no apparent adverse effects.

Consumption of fish from the bayou near the confluence with Rocky Branch presents a potential risk between  $10^{-3}$  to  $10^{-4}$  excess cancers. Further downstream the risk drops to  $10^{-4}$  to  $10^{-5}$ .

Other contaminants that were detected but were not quantified may add to the risk presented by media at the site.

In addition, many of these pathways are additive, for example a sewage treatment plant worker could also utilize the area for recreation, and consume fish from the Bayou. The potential risk from all exposure routes would have to be added to determine the cumulative risk.

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Appendix A  
METHODS FOR THE ENDANGERMENT ASSESSMENT

This appendix presents the detailed methods used in the endangerment assessment.

ESTIMATING HEALTH RISKS CAUSED BY CARCINOGENS

To estimate human health risks from carcinogens, the following information must be known or derived:

- o Lifetime average ingestion rates for soil, sediment, and fish
- o Chemical concentration in soil, sediment, and fish
- o Cancer potency

The calculation of risk from carcinogens is based on a lifetime average daily intake per kilogram of body weight. Because the possible ingestion of soil varies over a 70-year lifetime in relation to age and body weight, an age- and time-weighted average ingestion for soil is used. This accounts for the relatively higher ingestion rate rate per kilogram of body weight in the younger age classes.

The units on the carcinogenic potency factor estimates from the U.S. EPA Carcinogen Assessment Group (CAG) are (mg/kg body weight/day<sup>-1</sup>). The lifetime average chemical intake must be estimated, therefore, in terms of mg of carcinogen/kg body weight/day.

## SOIL INGESTION--RESIDENTIAL SETTING

The lifetime average sediment ingestion rate (LASI, in grams of sediment per kilogram of body weight/day) for residents was estimated from:

$$LASI = \frac{1}{N} \sum_{i=1}^N \frac{s_i}{b_i}$$

where:

- N = number of years in a lifetime (70)
- $s_i$  = soil ingestion in year i (gm/day)
- $b_i$  = body weight in year i (kg)

For a 70-year lifetime, the estimated LASI was as 0.028 gm/kg body weight/day based on the data in Table A-1. The derivation of this is demonstrated in Table A-2.

### Lifetime Average Chemical Intake

The lifetime average chemical intake from soil (LACIS) ingestion or the lifetime dose from soil ingestion is calculated with the LASI as follows:

The lifetime average chemical intake from soil ingestion, LACIS, is:

$$LACIS = LASI \times C_s \times f \times a$$

where:

- $C_s$  = soil or sediment chemical concentration
- f = fraction of year that exposure occurs
- a = fraction absorbed in the gut

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Table A-1  
ESTIMATED SOIL INGESTION  
BY BODY WEIGHT AND AGE

<u>Age (Years)</u>	<u>Body Weight (kg)</u>	<u>Estimated Ingested Soil/ Sediment<sup>a</sup> (g/day)</u>
0-0.75	5	0
0.75-1.5	8	1
1.5-3.5	12	10
3.5-5	15	1
5-18	38	0.1
>18	70	0.1

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<sup>a</sup>From Kimbrough et al., 1984

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Table A-2  
LIFETIME AVERAGE CHEMICAL INTAKE DERIVATIONS

LIFETIME AVERAGE SEDIMENT INTAKE DERIVATION (LASI)

(365.25 days x 0.75 yr) x 0 gm + 5 kg	=	0
(365.25 days x 0.75 yr) x 1 gm + 8 kg	=	34.242
(365.25 days x 2.0 yr) x 10 gm + 12 kg	=	608.75
(365.25 days x 1.5 yr) x 1 gm + 15 kg	=	36.525
(365.25 days x 13 yr) x 0.1 gm + 38 kg	=	12.954
(365.25 days x 52 yr) x 0.1 gm + 70 kg	=	<u>27.133</u>

719.145 gm/kg/70 yr

10.273 gm/kg/year

0.0281 gm/kg/day

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For the soil ingestion or inhalation route, the exposure duration represents the number of days that an individual will contact the contaminated soil or sediment. In a residential setting, behavior patterns and seasonal conditions will most influence the duration. Children and adults who enjoy playing outdoors and household pets could contact soil frequently. Cold or wet weather usually deters outdoor activities and decreases exposure. Similarly, dust generation and the resulting exposure is essentially eliminated when the soil is very wet or frozen.

Near Jacksonville, the temperatures are at or below freezing 71 days/year. Dust emissions are considered negligible on days when precipitation exceeds 0.01 inch (Kimbrough et al., 1984), which is reported to be an average of 103 days/year. (NOAA 1980). NOAA reports that approximately 80 percent of the precipitation days occur outside of the winter months. Thus, the area near the site has approximately 153 days/year (71 + 80 percent of 103) when climatic conditions would prevent outdoor activity and dust emissions.

Under these assumptions:

$$f = 1 - (153/365)$$

$$e = 0.58$$

and  $a = 0.2-0.3$  according to Schaum (1984).

$$LACIS = LASI \times Cs \times f$$

$$f = 1 - (295/365.25) = 0.2$$

$$LACIS = LASI \times Cs \times f$$

$$f = 1 - (295/365.25) = 0.2$$

Using the high end estimation for absorption of TCDD where:

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$$\begin{aligned}\text{LACIS} &= 0.028 \times 0.58 \times 0.3 \times \text{Cs} \\ &= 0.005 \times \text{Cs}\end{aligned}$$

### Risk Estimation

The excess lifetime cancer risk from soil ingestion in the residential setting is calculated as:

$$\text{Risk} = 1 - \exp (-P_i \times \text{LACIS}_i)$$

where:

$$P_i = \text{potency of carcinogen obtained from EPA CAG} \\ (\text{mg/kg-day})^{-1}$$

$$\text{LACIS}_i = \text{lifetime average chemical intake from soil} \\ \text{ingestion (mg/kg-day for chemical i)}$$

### Lifetime Average Chemical Intake Calculation

The lifetime average chemical intake from soil or sediment (LACIS, in milligrams of chemical per kilogram of day weight/day) ingestion is the lifetime exposure to contaminants in soil or sediment by ingestion.

The lifetime average chemical intake from soil or sediment ingestion (LACIS) is:

$$\text{LACIS} = \text{LASI} \times C_s \times f \times a$$

where:

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$C_s$  = soil or sediment chemical concentration

$f$  = fraction of year that exposure occurs

$a$  = fraction absorbed in the gut

For the soil or sediment ingestion or inhalation route, the exposure duration represents the number of days that an individual will contact the contaminated soil or sediment. In an occupational setting, work days and seasonal conditions will most influence the duration of exposure. The average work period is 243 days/year considering weekends (104 days), estimated vacations (10 days) and holidays (8 days). Cold or wet weather usually deters outdoor activities and decreases exposure. Similarly, dust generation and the resulting exposure is essentially eliminated when the soil or sediment is wet or frozen.

Near Jacksonville, the temperatures are at or below freezing 71 days/year. Dust emissions are considered negligible on days when precipitation exceeds 0.01 inch (Kimbrough, et al. 1984), which is reported to be average of 103 days/year. NOAA reports that approximately 80 percent of the precipitation days occur outside of the winter months. Thus, the area near the site has approximately 153 days/year when climatic conditions would deter outdoor activity and reduce dust emissions.

Under these assumptions:

$$\begin{aligned} f &= (1 - (153/365) \times (243/365)) \\ &= 0.39 \end{aligned}$$

and  $a = 0.2 - 0.3$  according to Schaum (1984).

Using the high end estimate for absorption of TCDD:

$$\begin{aligned}\text{LACIS} &= 0.00082 \times 0.39 \times 0.3 \times C \\ &= 0.00096 \times C\end{aligned}$$

### Risk Estimation

The excess lifetime cancer risk from soil or sediment ingestion is estimated as:

$$R_i = 1 - \exp (-[P_i \times \text{LACIS}_i])$$

where:

$R_i$	=	Lifetime excess cancer risk from chemical <sub>i</sub>
$P_i$	=	Carcinogenic potency factor obtained from EPA CAG (mg/kg-day) <sup>-1</sup>
LACIS	=	Lifetime average chemical intake from soil/sediment ingestion (mg/kg body/day) for chemical <sub>i</sub>

### SOIL/SEDIMENT INGESTION - RECREATIONAL SETTING

Three ingestion rates are estimated (1) for childhood exposure, (2) adult exposure, and (3) combined exposure.

Childhood exposure is based on:

- o 0.1 gram/day soil intake
- o Soil exposure 2 days per week, 6 months per year
- o Occurs from age 5-18
- o Body weight 38 kg
- o 70-year lifetime

Adult exposure is based on:

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- o 0.1 gram/day soil intake
- o Soil exposure 2 days per week, 6 months per year
- o Occurs from age 18-60
- o Body weight 70 kg
- o 70-year lifetime

Combined exposure is based on:

- o Combining the childhood and adult exposure
- o 70-year lifetime

This results in a LASI for childhood of 0.00007 g/kg/day, a LASI for adults of 0.00012 g/kg/day and a LASI for the combined of 0.000019 g/kg/day.

As in the other soil exposures they are limited by climatic conditions and absorption in the gut. Intake calculations are the same as in the occupational setting except the exposure fraction. In the recreational setting the exposure fraction is calculated as:

$$f = 1 - (153/365)$$

$$f = 0.58$$

#### FISH INGESTION - ADULT SETTING

##### Lifetime Average Ingestion Rates

The lifetime average fish consumption intake (LAFC, in g/kg body weight/day) for adults is estimated to be 0.056 from:

$$LAFC = \frac{n}{N} \times \frac{Si}{bi}$$

where:

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N = number of years in a lifetime (70)  
 n = number of adult years in lifetime between  
 ages 18 and 60 (42)  
 Si = fish intake (6.5 g/day)  
 bi = body weight of adult (70 kg)

The LAFC is estimated as 0.056 g/kg body weight/day.

### Intake Calculation

The lifetime average chemical intake from fish consumption (LACIF) is the lifetime exposure to contaminants from fish consumption.

The lifetime average chemical intake from fish tissue (LACIF), is

$$\text{LACIF} = \text{LAFC} \times A_F \times C_F = 0.9 \times C_f$$

where:

$$C_F = \text{fish tissue chemical concentration, mg/kg}$$

$$A_F = \text{absorption fraction in gut} = 0.9$$

### Risk Estimation

The excess lifetime cancer risk from fish tissue consumption is estimated the same as soil risk but substitute LACIF for LACIS.

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Appendix B  
CHEMICAL AND TOXICOLOGICAL PROPERTIES OF THE  
CHEMICALS OF CONCERN

INTRODUCTION

This section discusses the chemical and toxicological properties of all compounds that were analyzed for and detected for the RI.

- o 2,3,7,8-tetrachlorodibenzo-p-dioxin, (TCDD or dioxin)
- o chlorophenoxyherbicides; 2,4-D and 2,4,5-T
- o chlorinated benzenes
- o chlorinated phenols

CHEMICAL PROPERTIES

TCDD

There are theoretically 75 different chlorinated dibenzo-p-dioxin structures with varying degrees of chlorination. The most toxic dioxin that has been isolated and tested is 2,3,7,8-tetrachlorodibenzo-p-dioxin, commonly referred to as "TCDD" or simply "dioxin."

TCDD is inadvertently synthesized in concentrations of a few parts per million or less during industrial processing of chlorinated phenols, a widely used class of industrial organic chemicals. Elevated temperatures during chlorinated phenol processing have been shown to increase the formation of TCDD. TCDD is also generated during the combustion of

chlorinated phenols. Some research suggests that TCDD may form in refuse incinerators, fossil-fueled power plants, gasoline and diesel-powered motors, fireplaces, charcoal grills, and cigarettes; however, more research is needed to verify these findings.

It was not until 1957 that TCDD was synthesized and isolated from other chlorinated dioxin isomers. Since then, researchers have investigated various aspects of TCDD generation, environmental behavior, toxicity, and destruction. Most of this background data, especially the toxicity data, pertains to solubilized TCDD (a relatively mobile form), with only limited data applicable to TCDD complexed in soil. Several studies have indicated that the behavior of TCDD in the environment can change substantially depending on the nature of the TCDD-containing matrix.

TCDD is a highly symmetrical and stable nonpolar chlorinated organic compound that lacks chemically reactive functional groups. Pure TCDD is a colorless crystalline solid with an approximate melting point of 305°C and is believed to have a low vapor pressure of roughly  $10^{-6}$  mm of mercury at room temperature. It is commonly accepted that TCDD has low water solubility at room temperature, approximately 0.2 parts per billion. TCDD is categorized as a lipophilic compound, which means that it has a relative affinity for fats, waxes, and related organic compounds. TCDD solubilities have also been reported at 10 ppm in methanol, 40 ppm in lard, 570 ppm in benzene, and 1,400 ppm in ortho-dichlorobenzene.

This summary of TCDD behavior in the environment focuses on TCDD in soil. Several studies have indicated that the behavior of TCDD in the environment can change substantially depending on the nature of the TCDD-containing matrix. Research in the past 5 years has shown that TCDD binds with

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soil and becomes increasingly difficult to extract with time. This binding mechanism is not precisely understood, but is generally believed to be either a physical adsorption on soil particle surfaces or a chemical bonding with the soil humus fraction.

Leachate characterization studies conducted by the EPA have verified the soil binding phenomenon. In the studies, five TCDD-contaminated soil samples with TCDD concentrations ranging from 118 to 780 ppb were vigorously mixed with deionized water for 24 hours, decanted, and sequentially filtered to 0.45 microns. The filtrate samples were analyzed for their TCDD concentrations after each filtration stage. Of the five samples analyzed after the 0.45-micron filtration, four samples had nondetectable levels of TCDD. Duplicate analyses for the remaining sample showed TCDD concentrations of 6 ppt and 12 ppt. EPA is conducting additional leachability tests to determine more accurately TCDD's leachability from various soil matrices and to develop appropriate leachate treatment methods for TCDD contamination storage facilities.

The potential for TCDD in soil to leach into groundwater appears to be low due to its strong soil binding and its low solubility in water. Evaporation of TCDD is not well understood, but appears to be quite low, especially when in soil.

It appears that the primary mechanism for movement of TCDD in soil is displacement of contaminated soil particles. This can occur by wind or water erosion, contact with people, animals, or vehicles, or by intentional movement of the soil. In these ways, dioxin in soil may spread across an area and into the air or surface water.

It appears that plant uptake of TCDD has shown mixed experimental results that have not been resolved. However, several

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researchers believe that plants do not uptake TCDD that is bound in a soil matrix.

TCDD-contaminated soil is introduced into animals or humans through the respiratory tract, the skin, and the digestive tract. Recent animal research has shown that the absorption of TCDD into the body through the skin or the intestinal tract is reduced when it is mixed with soil. This absorption was found to further decrease as the time of contact between TCDD and soil was increased.

Studies have shown that TCDD in aquatic systems can be bioconcentrated in fish to approximately 30,000 times the surrounding water concentration. According to Dr. Stalling of the Columbia National Fisheries Research Laboratory, research indicates that TCDD in soil that is translocated into streams may be available at significant levels for fish uptake and concentration.

Biodegradation of dioxin in soil has not been demonstrated to any significant level. Although several previous studies indicated that appreciable biological degradation of TCDD in soil did occur, more recent studies have shown, and several researchers of previous studies have also concurred, that many of these results were likely due to inadequate analytical procedures and experimental controls.

Thermal degradation of TCDD in soil is essentially nonexistent in the environment. Pure TCDD is not appreciably decomposed by laboratory incineration at 700°C. TCDD bound to particulates has been said to resist thermal decomposition at incinerator temperatures up to 1000°C.

TCDD bound in soil does not exhibit any significant ultraviolet degradation. However, substantial ultraviolet

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degradation of TCDD will occur in several hours to a few days in the surface soil layers if TCDD is solubilized in a thin light-transmitting phase with a hydrogen donor present and exposed to sunlight or artificial ultraviolet light.

In summary, TCDD is very persistent. Natural environmental degradation does not occur for all practical purposes. This is evidenced by its presence at a constant level in the soils in Times Beach and other Missouri sites after more than 10 years.

#### CHLOROPHENOXY HERBICIDES

##### 2,4-Dichlorophenoxyacetic Acid (2,4-D)

2,4-D is a herbicide which is commonly used in its salt or ester forms. Agent Orange is a 50:50 mixture of 2,4-D and 2,4,5-T. It is a colorless, odorless white to yellow solid with a molecular weight of 221.04.

It has a melting point of 138°C and boiling point of 160°C at 0.4 mm Hg pressure. It is almost insoluble in water but soluble in alcohol and organic solvents. It does not readily degrade and is persistent in the environment. Physical and chemical characteristics are presented in Appendix Table B-1.

Mobility of 2,4-D is limited because of its insolubility in water. It is readily adsorbed by organic matter in soils and by clay particles in acidic systems. Primary means of environmental transport is through soil movement, i.e., surface runoff, dust entrainment, sediments. In sediments, 2,4-D is expected to be found in lower strata due to its specific gravity (>1). It is not expected to bioaccumulate in the food chain as it rapidly is excreted by animals.

### 2,4,5-Trichlorophenoxyacetic Acid (2,4,5-T)

2,4,5-T is a herbicide which was developed for use during World War II. It was discovered in 1955 that the contaminant TCDD associated with 2,4,5-T, caused chloracne in workers exposed to the compound. Despite this knowledge, use of the herbicide spread. It was used as a weed killer on range land, pastures, nursery and rice crops. In 1974 the US EPA banned use of 2,4,5-T on food crops. It was widely used in Vietnam from 1962 until 1969. It is a colorless to tan, odorless, noncombustible solid used as a liquid mix for herbicide. It has a melting point of 153°C and a vapor pressure of 0.0 mm Hg at 20°C. The compound is insoluble in water. 2,4,5-T acts in a manner similar to 2,4-D in the environment.

### CHLORINATED BENZENES

The chlorinated benzenes are a group of cyclic aromatic compounds in which one to six H atoms have been replaced with up to six chlorine substitutes. Twelve different compounds are possible. All have relatively low water solubility, with solubility decreasing with increasing chlorination, low to moderate vapor pressure, with vapor pressure decreasing with increasing chlorination and low flammability. Their octanol:water partition coefficient is moderate to high and increases with increasing chlorination. This means the higher chlorinated compounds, such as hexachlorobenzene, have a high tendency to adsorb onto sediments. All are considered to be volatile, except for hexachlorobenzene and all are only slightly reactive. The chlorinated benzenes are readily transported in the atmosphere. They are likely to enter the atmosphere as a result of volatilization and/or evaporation from soil or water. Once in the atmosphere they may be degraded by chemical or sunlight catalyzed reactions to

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nitro benzenes and/or nitrophenols. Atmospheric residence time is greater for the more highly chlorinated compounds. Chlorinated benzene compounds in the air may also become adsorbed onto particles that then settle or are removed from the atmosphere by rain. Transport of chlorinated benzenes in aqueous systems is limited. Most are readily evaporated from both aerated and nonaerated waters. In a controlled experiment greater than 99 percent of monochlorobenzene, 1,2-dichlorobenzene, 1,4-dichlorobenzene and 1,2,4-trichlorobenzene evaporated in four hours from aerated water and in 72 hours from nonaerated waters. The half-life of evaporation has been calculated using Henry's Law constant and assumptions on water depth, wind speed, ambient temperature, etc. Monochlorobenzene, 1,2-dichlorobenzene and 1,2,4-trichlorobenzene have calculated half-lives of 4.6 minutes, 8.1 minutes, and 0.75 hours, respectively.

The fate of chlorinated benzenes left in water has not been completely characterized. It is believed that microbial degradation occurs on the lower chlorinated compounds, but has yet to be demonstrated. All of the chlorinated benzenes have a medium to strong tendency to adsorb onto soils and sediments. This adsorption potential increases with increasing chlorination and increasing organic matter content of the soils and sediments. Once adsorbed, movement through soils is dependent on the soil type and temperature and the characteristics of the leachate. The potential for downward migration is high. Volatilization from porous soils to the atmosphere is a potential transport route. Again, this depends on soil type and temperature, each 10°C increase in temperature increases volatilization 3.5 times.

Vapor pressure decreases with increasing chlorination. The fate of chlorinated benzenes in soils and sediments is similar to that in water, only reaction times differ. Little

microbial degradation occurs in soils. Chlorophenols are the likely primary product of degradation. Chlorinated benzenes are persistent in soils once adsorbed, with persistence a function of chlorination (U.S. EPA 1980).

#### CHLORINATED PHENOLS

The chlorinated phenols are a group of compounds made up of a benzene ring to which one hydroxy (OH) group and one to five chlorine atoms are attached. Nineteen different compounds are possible. All compounds have low water solubility, low vapor pressure and low flammability. The chlorophenols of 2-chlorophenol, 2,4-dichlorophenol and 2,4,6-trichlorophenol are weakly sorbed to soil particles and move readily through the soil profile. These may undergo photochemical decomposition in the aqueous phase or in surface soils. Biodegradation is an important fate of these compounds from soils. Microbial decomposition occurs rapidly and bioaccumulation does not occur to any degree. Pentachlorophenol (PCP), however, does not follow in the same manner as the other chlorophenols. PCP is tightly bound to soil particles, is not volatilized from an aqueous environment and is persistent in surface soils. Microbial decomposition does not occur to any degree.

#### TOXICOLOGICAL PROPERTIES

##### TCDD

Exposure to TCDD is possible through ingestion of food or water, inhalation, or dermal application. Because TCDD is strongly sorbed to soils and sediment, drinking water contamination is very unlikely, especially in groundwater supplies. Surface waters can be contaminated from contaminated industrial effluents or washouts from contaminated disposal

sites, however, even in these cases, TCDD was found to be strongly sorbed to sediments and biota (U.S. EPA 1984a).

Possible TCDD contaminated food items can include plant crops sprayed with weed killing herbicides such as Silvex and 2,4,5-T, livestock raised on TCDD contaminated forage and other organisms that have bioconcentrated the chemical through the food chain. Studies on animals indicate that TCDD is readily absorbed through the gastrointestinal tract, but there is little evidence that TCDD is taken up or absorbed in food crops (U.S. EPA 1984a). Research indicates that when TCDD is a contaminant in an organic herbicide, rapid photochemical degradation occurs during the application process (Crosby 1983).

TCDD can bioaccumulate in aquatic and terrestrial organisms. Bioconcentration studies using a variety of fish species have resulted in measured bioconcentration factors (BCF). BCF relates the concentration of a chemical in aquatic species to the concentration of the chemical in water. Bottom feeders, carnivores, and species with high fat content had higher BCF values. These species include catfish, carp, trout, and salmon. Calculated BCF values using the octanol:water partition coefficient resulted in a range of 7,000 to 900,000 (U.S. EPA 1984a). Currently, the U.S. EPA's best estimate for the BCF of TCDD in aquatic organisms is 5,000. Recent studies by EPA ORD indicate dioxin half-life in fish to be approximately one year. (Ecology USA 1985). TCDD also apparently accumulates in fat tissue of cattle grazing on contaminated pasture (Kimbrough 1983). Levels of TCDD in cattle were found to range from 4 to 70 parts per trillion (ppt). TCDD can also be found in mammalian milk.

Inhalation of TCDD can occur during agricultural spraying, industrial incineration, industrial accidents, or as dust

borne particles. No analytical information is available concerning effects of inhaling TCDD (U.S. EPA 1984a).

Dermal exposure to TCDD is most likely to occur during the manufacture and application of contaminated chlorinated herbicides. Many people were exposed to dioxins from the application of Agent Orange during the Vietnam war. Researchers have attempted to quantify human health effects from possible exposure to TCDD by studying health histories of manufacturing and agricultural workers and Vietnam veterans most likely exposed to this chemical. The most common and obvious effect reported was chloracne, or skin lesion. Other possible effects are included in the following discussions of acute and chronic toxicities, mutagenicity, teratogenicity, and carcinogenicity.

After absorption of 2,3,7,8-TCDD in most animal species studies, this chemical is most often found in the liver and adipose tissues. Biological metabolites of TCDD are not considered to be toxic compared to TCDD itself. Elimination routes for TCDD in mammals include lactation, direct intestinal elimination, and expectorate.

The toxic effects of TCDD have been extensively studied in animals. These studies indicate that the compound is toxic from both an acute and chronic standpoint. Animal studies indicate that TCDD is a potent hepatic enzyme inducer in most species. Beside the liver, other target organs include the thymus, testicles, spleen, gall bladder, skin, and urinary tract (Olie, et al. 1977). Of special interest from an acute toxicity standpoint is the wide species variation. The oral LD50 values for TCDD range from 0.6  $\mu\text{g/kg}$  body weight for the guinea pig to 5,051  $\mu\text{g/kg}$  body weight for the hamster. Toxicities vary with age, sex, and strain of test animal, with young animals more susceptible than older animals.

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Differences between animals tested showed no apparent trend (U.S. EPA 1984a). The most common toxic responses included loss of body weight, thymic trophy, and increase in liver weight. Often toxic responses were delayed.

Chronic effect studies indicate that TCDD exposure may produce chloracne, liver damage, kidney damage, immunological alterations, hematological alterations, gastrointestinal tract changes, and neuropsychiatric effects in test animals. Subchronic effects reported in rats include lethargy, decreased body weights, liver pathology, biochemical evidence of liver damage, thymic atrophy, decreased lymphatic tissues, disturbance of porphyrin metabolism, slight alterations in the hematopoietic system and mild adverse effects on both male and female reproductive systems. Long recovery times were seen in subchronic studies (U.S. EPA 1984a). Subchronic effects were observed in humans after the accidental exposure to TCDD in Seveso, Italy. Over 200 cases of chloracne were reported with the most severe fully recovered after 18 to 24 months (with one exception). Other symptoms included signs of liver damage, raised serum transaminase and glutamyl transferase, and some neurological effects (U.S. EPA 1984a). Before chloracne appears, overexposure may be indicated by burning sensations in the eyes, nose, and throat, headache, dizziness, and nausea. Other symptoms include joint pain, fatigue, insomnia, irritability and nervousness. Emotional disorders, difficulties with muscular and mental coordination, blurred vision, and loss of taste or smell may occur. Deaths related to TCDD induced liver damage have occurred (Olie, et al 1977).

Animal studies also indicate that TCDD is a developmental toxin. It acts as a teratogen in mice and hamsters, but not primates. TCDD produces fetotoxicity at doses higher than doses causing teratogenic effects. The most common

teratotoxic responses in rats and mice are increased cleft palate and kidney abnormalities. TCDD appears to be acting synergistically with 2,4,5-T with respect to the increase in cleft palate occurrences. Many attempts have been made to link fetotoxicity and teratotoxicity to TCDD or herbicide exposure in pregnant women. Studies thus far have not been statistically conclusive regarding any toxic effects in human reproduction (U.S. EPA 1984a). Animal testing and human health histories have shown no increase in fetotoxicity or teratotoxicity resulting from male exposure to TCDD.

Genotoxicity testing of TCDD has been extensive, however, the results of these studies have indicated little potential for mutagenic effects (U.S. EPA 1984a). While some studies indicate that TCDD may be a bacterial mutagen and cause cytogenetic damage (Olie, et al. 1977), overall, the data indicate little potential for the interaction of TCDD with nucleic acids or the ability of TCDD to produce chromosomal aberrations (U.S. EPA 1984a).

Several animal studies using TCDD have indicated an oncogenic effect. This effect has been seen in both mice and rats. Also, studies have been performed that have led to the conclusion that TCDD is a tumor promoter. Recently, the National Toxicology Program (NTP) bioassay program concluded that TCDD is a carcinogen when studied in rats and mice (NTP 1983). Kolbye (1983) states that dioxins are secondary carcinogens only able to promote tumors in already initiated cells after continuous, consistent, and selective pressures on that cell.

Researchers have tried to link human cancers of various types to TCDD exposure. The most significant association appears to be between TCDD and soft tissue sarcoma. The most recent studies prepared by the U.S. Air Force involving 1,247 people exposed to TCDD in Vietnam showed no significant cancer

increase. Small sample size or inadequate study plans have discounted many human health surveys attempting to link cancer to TCDD or herbicide exposure.

TCDD is suspected of being a human carcinogen because of multiple positive animal carcinogenicity studies. The U.S. EPA's position is that there is no recognized safe level for a human carcinogen and the recommended concentration in water for maximum protection of human life is zero. Because attaining a zero concentration may not be feasible at this time, the concentrations corresponding to incremental increased lifetime cancer risk levels have been estimated (U.S. EPA 1984a) and are shown below:

Exposure	Concentration of TCDD in water resulting in $10^{-6}$ incremental increase for lifetime cancer risk ( $\mu\text{g/L}$ )
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Consumption of 2 L drinking water per day and 6.5 g of fish and shellfish.	$1.3 \times 10^{-8}$
--	----------------------

Consumption of 2 L drinking water per day only	$2.2 \times 10^{-7}$
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As can be seen in this example, the water contamination level must be 17 times as high for the same risk level as when no contaminated fish are consumed.

The Center for Disease Control has recommended that residential soils contain TCDD at average concentrations no greater than 1 ppb. Higher levels in commercial areas may represent an acceptable risk level to non-occupationally exposed persons (Kimbrough 1983). The FDA has recommended against

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consumption of food containing TCDD at levels greater than 50 ppt or more than twice per month at greater than 25 ppt. No tolerance levels have been established for TCDD on food crops (Miller 1983).

In summary, TCDD is considered to be an unusually toxic compound with demonstrated acute, subchronic, and chronic effects in man and animal. Reported adverse effects include chloracne and damage or changes to the liver, nervous system, immune system, and reproductive system. Special groups at risk are those employed in the manufacture of chemicals which may contain 2,3,7,8- as a contaminant, women of child-bearing age, and especially the fetus.

#### CHLOROPHENOXY HERBICIDES

2,4-dichlorophenoxy acetic acid and 2,4,5-trichlorophenoxy-acetic acid are used in several hundred commercial formulations, and sometimes combined in mixtures, e.g., Dacamine 2D/2T.

These compounds and their salts and esters are moderately irritating to the eyes, skin, respiratory and gastrointestinal tract. Some of these compounds, such as 2,4-D, can enter the body through the skin. They also are absorbed from the lungs and through the gastrointestinal tract. Generally, they are considered to be of low to moderate toxicity. They do not remain stored in fat to a great degree, and are excreted within hours, or at the most, within days. There have been a limited number of reports in medical literature of toxic effects in humans from exposure to 2,4-D. Three cases of peripheral neuritis were reported for workers occupationally exposed to 2,4-D. Local depigmentation in some individuals has been attributed to prolonged and repeated

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dermal contact with chlorophenoxy compounds (NIOSH 1978, Morgan 1982).

Single doses of 5 mg/kg of body weight of 2,4-D and 2,4-T were administered to human subjects without adverse effects. No adverse effects were seen in one person who consumed 500 mg of 2,4-D per day for 3 weeks (Morgan 1982).

The signs and symptoms of acute poisoning from ingestion of large amounts of chlorophenoxy compounds are irritation of the mouth, throat and gastrointestinal tract, spontaneous emesis, chest pains (due to esophagitis), abdominal pain, and diarrhea. Injury of the gastrointestinal tract usually does not progress to ulceration and perforation. Once compounds are absorbed they can cause fibrillary muscle twitching, skeletal muscle tenderness, and myatonia (stiffness of extremities). Symptoms associated with ingestion of very large amounts of chlorophenoxy acids are metabolic acidosis, fever, tachycardia, hyperventilation, vasodilation and sweating. Some cases have been characterized by coma and convulsions (Morgan 1982).

The animal toxicity of 2,4-D and 2,4,5-T is similar. The low cumulative effect of these compounds has been demonstrated in feeding studies where animals tolerated repeated exposures to doses slightly smaller than the single toxic dose (Cassarett and Douell 1980).

Based on animal studies done by Rowe and Hymas, it was concluded that 2,4-D had a low chronic toxicity. For 2,4-D, oral LD 50 values for several animal species ranged from 100 to 1,000 mg/kg. For 2,4,5-T, oral LD 50 values ranged from 300 to 1,000 mg/kg for several species. Dogs were found to be more sensitive than other animal species, with an LD 50 of 100 mg/kg for 2,4,5-T isopropylestec (NIOSH 1978). In

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animal experiments, large doses of 2,4-D caused vomiting, diarrhea, anorexia, weight loss, ulcers of the mouth and pharynx, and injury to the kidney and liver, and central nervous system. Muscular effects have been seen in some species, specifically myatonia, or stiffness of the extremities, which was apparently due to CNS damage. Also, in heavily dosed animals, demyelination was observed in the dorsal columns of the cord, and EEG changes indicated functional disturbances in the brain.

#### 2,4 Dichlorophenoxyacetic Acid (2,4-D)

Based on a literature review, EPA concluded in 1980 that the acid form of 2,4-D was of low to moderate toxicity, and does not pose an imminent health hazard when used properly. Based on available studies, 2,4-D is not known to be carcinogenic in animals or humans. Results of mutagenicity tests have been mixed; the majority of results have been negative, while there have been three positive results reported. Reproductive studies conducted on mice, rats and hamsters showed slight fetotoxic effects, at lower dose levels, including edema (swelling of tissues). Very high dose levels caused skeletal malformations and cleft palates.

#### 2,4,5-Trichlorophenoxyacetic Acid

The mammalian toxicity of 2,4,5-T is low. It is slightly irritating to the skin, and overexposure to this compound may cause abdominal pain, nausea, vomiting, diarrhea and blood in the stool. Dioxin isomers are unwanted contaminants in 2,4,5-T, especially the extremely toxic isomer, 2,3,7,8-TCDD. Chloracne seen in industrial workers who worked in a 2,4,5-T manufacturing facility, was attributed to the TCDD contaminants (2,3,7,8-TCDD or 2,3,6,7-TCDD).

Several incidents of human exposure to 2,3,7,8-TCDD have involved the 2,4,5-T herbicide compound. In March 1979, the EPA declared an emergency suspension of 2,4,5-T and Silvex [s-(2,4,5-trichlorophenoxy) propionic acid], because of a reported increase in miscarriages in humans in Alsea, Oregon, an area where the herbicides were sprayed. These effects were believed to be a result of exposure to the dioxin contaminant in 2,4,5-T, and possible contamination of Silvex, because of its similarity to 2,4,5,-T. The defoliant, Agent Orange, used during the Vietnam War, is a 50:50 mixture of 2,4-D and 2,4,5-T, and is alleged to have caused serious medical problems in many Vietnam war veterans. This conclusion is contradicted by other studies.

Long term exposure to dioxin contaminants in 2,4,5-T may cause chloracne and liver damage. Hepatic toxicity resulting from exposure to 2,3,7,8-TCDD has been demonstrated in animal studies, and has been observed in human workers after industrial exposure. Teratogenic, embryotoxic and fetotoxic effects have been produced in animals exposed to 2,4,5-T containing 2,3,7,8-TCDD, and have been attributed to the teratogenic and fetotoxic potential of 2,3,7,8-TCDD.

Some positive results have been reported on mutagenicity tests done on 2,4,5-T (NIOSH 1983). 2,4,5-T is not known to be carcinogenic in humans or animals.

#### CHLORINATED BENZENES

Chlorinated benzenes consist of any benzene compound which contains one or more chlorine atoms. They range from the monochlorinated material to the hexachlorinated one. The dichloro, trichloro, and tetrachloro species can exist in various isomeric forms. There are several generalizations which can be made about these compounds which help

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understand their environmental qualities. In general, they are used as chemical intermediates for the synthesis of other compounds and can be used as pesticides. They may bioaccumulate in the environment with their potential for bioaccumulation increasing with chlorine content. As their chlorine content increases, their potential for biomagnification also increases, while their potential for biodegradation decreases (Sittig 1980). Volatility and adsorbability to organic sediments also increase with the number of chlorine atoms (U.S. EPA 1981). Acute toxicity generally decreases as the number of chlorine atoms increase. Most chlorinated benzenes are irritating to the skin, eyes, lungs, and mucous membranes (Sittig 1980).

Animal studies and human experience indicate that all isomers attack the liver, kidney, and nervous system as target organs for toxic effects. Often chlorobenzenes affect the reproductive systems (Clayton and Clayton 1981). Several representatives of the chlorinated benzene family will be discussed in the following pages.

Dichlorobenzenes have similar toxicities. Kidney, liver, lung, and the blood forming organs appear to be the primary target organs for toxicity based on human exposure information. They also may irritate skin, eyes and mucous membranes (Clayton and Clayton 1981). An NTP bioassay for carcinogenicity on the ortho isomer concluded that there was no evidence of carcinogenicity. The available mutagenicity data also indicate that this effect is of little concern. The usual route of exposure is inhalation or dermal contact, however, occasionally ingestion of the para isomer has been reported (Clayton and Clayton 1981). Trichlorobenzenes are produced in relatively small amounts. As with dichlorobenzenes, these isomers have similar toxicities. Human experience indicates that the target organs include lung, blood

forming organs, and skin. Based on animal study results, it may be concluded that the kidney, liver, adrenals, and nervous system are potential target organs. These compounds also may cause dermal, eye and mucous membrane irritation. One chronic study for carcinogenicity was negative; however, these results are not conclusive enough to give an indication one way or the other regarding potential carcinogenicity. Mutagenicity and teratogenicity testing results have been negative.

Although acute toxicity in animals has not been shown to be a major concern, (rat oral LD<sub>50</sub> 3500 - 10,000 mg/kg), human overexposure to hexachlorobenzene has resulted in a condition called porphyria cutanea tarda, an illness in which porphyrin metabolism is disturbed. This condition does not appear to be an occupational problem even among workers producing hexachlorobenzene (IARC 1979). Major outbreaks of this illness have been reported when humans ingest seeds treated with hexachlorobenzene. One outbreak involved 5,000 people. The illness was characterized by skin lesions, usually appearing in areas exposed to sunlight. Often the lesions became ulcerated and crusted. Other clinical symptoms included excessive hair growth and hyperpigmentation, corneal opacity, and liver damage. Even 20 years later, a few of the exposed individuals suffer from the hexachlorobenzene exposure. Young children appeared to be most sensitive to the effects of hexachlorobenzene and there were fatalities among this group (Clayton and Clayton 1981).

Animal toxicity studies appear to support human observations with regard to hexachlorobenzene induced toxicity. Like humans, long-term animal exposure to hexachlorobenzene resulted in porphyrin metabolism changes, liver toxicity, and parent to offspring transfer of hexachlorobenzene via mothers' milk. Animal studies also indicated there were effects on

the kidney, nervous system, and reproductive process (IARC 1979). Data are insufficient to assess the mutagenic potential of hexachlorobenzene.

Several chronic animal studies indicate that hexachlorobenzene is an animal carcinogen. Chronic oral exposure to hexachlorobenzene of rats, hamsters and mice have resulted in increased tumor incidence. Sites where incidents are elevated include liver, thyroid, and blood vessels (NTP 1983).

Chlorobenzenes are listed by several organizations concerned with human health effects of these compounds. Recommendations for occupational air levels of exposure to several chlorobenzenes are as follows: chlorobenzene - 75 ppm; o-dichlorobenzene - 50 ppm; p-dichlorobenzene - 75 ppm; and 1,2,3-trichlorobenzene - 5 ppm (ACGIH 1985). These values are for 8 hours/day, 5 days/week exposure to airborne concentrations of these materials. The U.S. EPA has set the following criteria for chlorobenzenes in drinking water: monochlorobenzene 20 µg/l; dichlorobenzenes 230 µg/ml; trichlorobenzene 13 µg/l; tetrachlorobenzene 17 µg/l; and pentachlorobenzene 0.5 µg/l (U.S. EPA 1980). The mono and trichlorobenzene values are based upon odor and taste, the others on toxicity. The U.S. EPA states that they feel that exposure levels for carcinogens ought to be zero; however, EPA has set levels of incremental risk for carcinogen exposure. For hexachlorobenzene, incremental risks of cancer of  $10^{-5}$ ,  $10^{-6}$ , and  $10^{-7}$  are 7.2 ng/l, 0.72 ng/l and 0.072 ng/l respectively (U.S. EPA, 1980). These values assume ingestion of 2 liters of water per day and consumption of 6.5 g of fish daily. The World Health organization has set a conditional daily intake for this compound at 0.5 µg/kg/day in foods (Clayton and Clayton 1981).

Since chlorobenzene compounds are irritating, toxic to the liver, and in general may adsorb to organic matter, care should be taken to avoid dermal contact with them. Many of these compounds are quite volatile and potential exposure may occur if sediments onto which they are entrapped are disturbed. Consideration ought to be given to respiratory protection if this is a possibility.

#### CHLORINATED PHENOLS

The degradation of herbicides 2,4-dichlorophenoxyacetic acid and 2,4,5-trichlorophenoxyacetic acid to chlorophenol isomers is an important source of human exposure to these compounds in the environment. Other sources of exposure are (1) from chlorination of phenols present in natural water and in effluents from waste treatment plants, (2) direct addition of chemicals from industrial sources, (3) wet and dry atmospheric fallout.

2-Chlorophenol is used as a starting material for higher chlorophenol isomers. Synthesis of herbicides 2,4-D and 2,4,5-T involve 2,4-dichlorophenol and 2,4,5-trichlorophenol as intermediates. Pentachlorophenol has been used for several decades as a wood preservative and fungicide. It is ubiquitous in the environment; low levels have been detected in sewer water, municipal water supplies, human food stuffs, and in blood, urine and fat of nonoccupationally exposed persons (U.S. EPA 1979).

Generally, the toxicity of chlorophenols to higher organisms is greater as the degree of chlorination of the isomer increases. However, there are exceptions; based on LD 50 values, 2,4-dichlorophenol and the trichlorophenols (2,4,5 and 2,4,6) are less toxic than 2-chlorophenol, and 2,4-dichlorophenol

is less readily absorbed through the skin. Pentachlorophenol is the most toxic of the chlorophenol isomers.

Information on effect of long-term exposures and chronic toxicity in humans of chlorophenol compounds has not been documented. The extremely poisonous compound 2,3,7,8-tetrachlorodibenzo-p-dioxin is a known impurity in technical grade formulations of chlorophenols, and may be responsible for reported industrial cases of chloracne.

All the compounds under discussion, 2-chlorophenol, 2,4-dichlorophenol, and pentachlorophenol are not known to be carcinogenic in humans or in laboratory animals. Some positive results from mutagenicity tests have been reported for isomers of 2-chlorophenol.

#### 2-Chlorophenol and 2,4-Dichlorophenol

2-chlorophenol is readily absorbed through the skin, whereas 2,4-dichlorophenol is not. Both compounds are readily absorbed from the gastroenteric tract. These compounds cause convulsions in high concentration with decreased activity and motor weakness. The convulsant action is most likely to be an undissociated molecule.

#### Pentachlorophenol

Pentachlorophenol is a compound which has been used for many years primarily as a wood preservative. The acute toxicity of this compound is very high. Commercially produced pentachlorophenol may be contaminated with dioxin compounds which are more toxic than pentachlorophenol. (NIOSH 1983).

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The acute toxicity of pentachlorophenol is high (oral LD 50 for rats, 50 mg/kg; oral LD 50 for rabbits 70 mg/kg; oral LD 50 for hamsters, 168 mg/kg).

Chronic effects from continuous exposure to low doses have not been clearly demonstrated in experimental animals. Sub-chronic and chronic feeding studies have been done in several mammalian species. In a 90 day feeding study with two groups of male rats fed pure pentachlorophenol (containing low levels of chlorodibenzo-p-dioxin contaminants) and technical grade pentachlorophenol (containing relatively high levels of these contaminants) at doses equivalent to approximately 50 mg/kg body weight per day, pathologic changes in the liver were seen when examined microscopically. A dose related decrease in calcium deposits in the kidneys of rats given dietary doses of pentachlorophenol at 25, 50 and 200 mg/kg body weight for 12 weeks was seen. (U.S. EPA 1979).

The compound has been shown to cause adverse reproductive effects in rats.

Pentachlorophenol is a highly toxic compound which is readily absorbed through the skin. Acute systemic toxicity in humans can occur following absorption through the respiratory tract, gastrointestinal tract and skin. This compound is rapidly absorbed through the gastrointestinal tract following ingestion. In cases of severe or fatal poisonings, symptoms include loss of appetite, respiratory difficulties, anesthesia, hyperpyrexia, sweating, dyspnea and rapidly progressive coma. Many cases of human intoxication have been reported, most of which involved direct absorption of pentachlorophenol or its sodium salt through the skin. Cases of pentachlorophenol poisoning which resulted primarily from inhalation of vapors or dusts have been reported as well. Acute poisoning from pentachlorophenol centers in the circulatory system and is

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accompanied by heart failure. (ACGIH 1979). The physiologic injury which results from poisoning is mainly vascular. Pentachlorophenol dust and mist cause irritation of the eyes and upper respiratory tract; absorption results in an increase in metabolic rate and hyperpyrexia; prolonged skin exposure causes an acneform dermatitis. Human exposure to dust or mist concentrations greater than  $1 \text{ mg/M}^3$  causes pain in the nose and throat, and violent sneezing and cough;  $0.3 \text{ Mg/M}^3$  may cause some nose irritation. Persons who work routinely with pentachlorophenol may have some tolerance to these respiratory effects, and may tolerate airborne concentrations up to  $2.4 \text{ mg/M}^3$ . Systemic intoxication is cumulative and has been fatal. Intoxication is characterized by weakness, anorexia, weight loss, and profuse sweating; there also may be headache, dizziness, nausea, vomiting, dyspnea, and chest pain. In fatal cases, the body temperature is frequently extremely high and death has occurred as early as 3 hours after the onset of symptoms. Other effects which may result from repeated exposure to pentachlorophenol are acneform dermatitis, bronchitis, and liver damage. (NIOSH 1983).

Minimum lethal concentrations for pentachlorophenol in air have not been defined. The threshold limit value, which is based on an 8 hour time weighted average exposure is  $0.5 \text{ mg/M}^3$  (ACGIH 1977). The risk of intoxication via inhalation is greater during hot weather; although the vapor pressure of PCP is low ( $.00011$  to  $0.12 \text{ mmHg}$  at  $20^\circ\text{C}$  to  $100^\circ\text{C}$ ) toxic levels of vapor can build up in hot, enclosed areas.

The exact dosage which produces illness in humans is not known. An oral lethal dose in humans of  $20 \text{ mg/kg}$  has been reported. Symptoms of poisoning occur at concentrations of  $40$  to  $80 \text{ mg/liter}$  in the blood. In fatal cases, blood levels

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have ranged from 46 to 156 mg/liter, and urine levels from 28 to 520 mg/liter (U.S. EPA 1979).

For acute intoxications, the urine pentachlorophenol concentration is frequently higher than the blood level. In humans and animals, pentachlorophenol is excreted primarily through the urine. One study suggested a ratio of 1.5 to 2.5 of pentachlorophenol in blood to pentachlorophenol in urine in humans (U.S. EPA 1979). Initial urinary elimination following exposure to pentachlorophenol may be rapid, but return to background levels may take a month or longer. Approximately 50 percent of the body load is excreted in the urine in 24 hours, and 70 percent to 80 percent is excreted in 4 days (U.S. EPA 1979). Renal competency, that is, the capacity of the renal system to handle the pentachlorophenol load, appears to be a factor in the extent of individual susceptibility to pentachlorophenol poisoning (U.S. EPA 1979).

Long-term chronic effects from exposure to low levels of pentachlorophenol have not been seen in humans. Low background levels of PCP have been found in the blood and urine of occupationally and non-occupationally exposed persons, but chronic effects from these levels have not been reported. A reversible effect on the kidney has been seen, where PCP exposure caused a decreased creatinine clearance and phosphorous reabsorption in the kidney. These effects were seen in workers chronically exposed to PCP in the wood treatment industry. Wood treaters were tested before, during, and after vacation, and significant differences were seen in blood and urine phosphorous levels and in creatinine clearance. In one study it was found that workers continuously exposed to PCP had elevated levels of gamma-mobility C-reactive protein in the serum. Elevated C-reactive protein levels are associated with inflammatory disorders and tissue damage,

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and it was inferred that PCP exposure may produce inflammation or tissue or damage (U.S. EPA 1979).

No evidence exists that pentachlorophenol is carcinogenic in humans and animals.

This compound does not appear to be teratogenic in rats. However, embryotoxic and fetotoxic effects have been observed in experiments with rats. Developmental effects were observed when high doses of pentachlorophenol were administered to maternal rats, which could have been due to direct toxic effect on the maternal rat, as placental transfer of the compound is minimal (U.S. EPA 1979).

Some positive results have been reported from mutagenicity testing of pentachlorophenol (NIOSH 1983).

#### PREVIOUS DIOXIN CASE STUDIES

Since 1949, more than 23 major industrial accidents involving solubilized dioxin have been reported around the world. In addition, many people have been exposed to solubilized dioxin in the chemical manufacturing industry and through transportation accidents, herbicide applications, waste handling, and chemical laboratory work. The following paragraphs summarize some of the major reported dioxin-exposure incidents and the public response to these hazards.

Seveso, Italy. On July 10, 1976, an accident at the Givaudan Factory ICMESA 2,4,5-TCP plant released a dioxin-contaminated chemical cloud that showered down on a farmland area of about 6,000 acres, inhabited by approximately 40,000 people. Thousands of animals died and approximately 134 people in the three designated zones of contamination developed chloracne (a skin disorder believed to be the most sensitive indicator

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of dioxin exposure in humans; it is most prevalent on the face, neck, and arms).

Some of the initial remedial measures implemented at Seveso included collecting and isolating contaminated materials, evacuating residents in contaminated areas, implementing measures to avoid contact with contaminated materials, and decontaminating affected materials. Numerous measures were studied for ultimate destruction of the contaminated material, including incineration, ultraviolet degradation, biological degradation, and chemical destruction. None of these options were considered viable for effectively treating the large volumes of contaminated soil. Removing the contaminated materials to offsite disposal site(s) was not considered acceptable either. Onsite disposal of contaminated soil in secured basins was considered the most practical option. This method has been used for disposing of the soil in the more highly contaminated zones.

Missouri Horse Arenas. In 1971, waste oils containing TCDD were sprayed for dust stabilization in three east-central Missouri horse arenas. In the most affected arena, 54 out of 57 horses exposed to the waste oil developed similar illness symptoms and died. Dead birds, cats, dogs, rodents and insects were also found in and around the arena. A 6-year-old girl who played regularly in the arena became very sick and lost 50 percent of her body weight. Five years later, she had apparently recovered.

The arena co-owners and their 10-year-old daughter have experienced severe headaches, nausea, and other symptoms. Human illness and animal illnesses and deaths occurred at the other two horse arenas. All three horse arenas were sprayed within one month of each other with waste oil from Bliss Waste Oil Company. That same company later sprayed

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dioxin-contaminated oil on unpaved streets in Times Beach. The source of the TCDD-contaminated oil was traced to the Northeast Pharmaceutical and Chemical Company (NEPACCO) in Verona. Analysis of a distillation residue, similar to that mixed in with the waste oil, revealed the residue contained 306 to 356 ppm TCDD. The Shenandoah Stables horse arena may have been sprayed with undiluted sill bottoms.

Soil from one of the arenas was later analyzed, and contained 31 to 33 ppm TCDD. The soil from each of these arenas was excavated and landfilled at various other Missouri sites. No further TCDD-related animal deaths or human illnesses have been reported since the soils were excavated.

Syntex--Verona, Missouri. In 1974, Syntex Agribusiness discovered a tank containing 4,300 gallons of distillation bottoms sludge contaminated with 343 ppm dioxin. The sludge was a byproduct formed in the production of hexachlorophene by NEPACCO, the previous facility occupants. Syntex implemented initial measures to provide security and safety for the storage tank and began investigating alternatives for destroying the dioxin, including treatment in a high-pressure reactor, incineration, treatment with a chemical process, and ultraviolet photolysis.

Other dioxin contamination has been discovered and is suspected around the Verona facility. This site is currently on the National Priority List.

Denney Farm Site, Missouri. In 1979-80, a trench at the Denney Farm site in southwest Missouri was found to contain approximately 90 barrels of dioxin-contaminated wastes disposed of by NEPACCO. Syntex Agribusiness, the EPA, and the Missouri Department of Natural Resources developed and implemented a cleanup program. The drums and soils

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contaminated from drum leakage were excavated and placed into storage in onsite concrete vaults at a cost of over \$1.8 million. The contaminated materials currently await final disposal or destruction.

Neosho Digester--Neosho, Missouri. A digester at the Neosho municipal wastewater treatment plant was used to store wastewater from NEPACCO during 1970 and 1971. In 1977, during a plant renovation, this digester was filled with gravel, soil, and debris. The digester overflowed and contaminated the ground nearby. The contaminated soil was excavated and put in a trench near the digester.

The EPA in 1981 sampled the digester and found concentrations as high as 60 ppb of TCDD and 1,000 ppm of TCP. In June 1984, the EPA issued an administrative order to the City of Neosho to cap the site and monitor groundwater.

Neosho Tank Spill Site--Neosho, Missouri. The Neosho Water and Wastewater Technical School conducted treatability studies on NEPACCO wastewater. The tank used to store the wastewater leaked onto the ground.

In 1981, the EPA found a TCDD concentration of 1.9 ppm in samples of the tank's contents. The highly contaminated soil next to the tank was excavated and put in 15 drums. The tank and drums were put in an Army ammunition bunker on the school property. The tank site was capped and fenced.

An EPA administrative order, issued in August 1981 and amended in June 1984, requires proper disposal of the drums and soil from the tank area. This waste was scheduled to be destroyed in EPA's mobile incinerator in the fall of 1984 but did not occur.

Rail Accident--Sturgeon, Missouri. In 1979 a tank car of orthochlorophenol contaminated with 37 ppb dioxin was derailed in Sturgeon, Missouri. Two workers who were involved in cleanup operations were later found to have low levels of dioxin in their blood. In 1982, \$57 million in damages were awarded to 75 workers exposed to dioxin contamination during this cleanup operation. This award was recently repealed and the suit is now awaiting further court action.

Union Carbide--Sydney, Australia. In 1978, dioxin wastes generated by Union Carbide in trichlorophenol manufacturing processes were discovered in garbage sites in three Sydney suburbs. The Australian government implemented a massive health record review of residents in proximity to these sites. This review apparently did not reveal any definite health problems associated with these landfills. It appears that no further remedial actions were implemented.

Agent Orange. Dioxin was found to be a contaminant in Agent Orange, a defoliant used in Vietnam. At least 4,800 veterans have asked for treatment because of their exposure to the herbicide, and considerable litigation has resulted from these incidents. An out-of-court settlement was recently reached between the veterans and several manufacturers of Agent Orange.

In 1977 more than 2 million gallons of Agent Orange were incinerated in the Pacific Ocean on the incinerator ship Vulcanus. Calculations on the dioxin combustion efficiency showed it exceeded 99.9 percent.

Activated coconut charcoal has been used by the Air Force to reduce the TCDD level from 8 ppm to 0.1 ppm in Agent Orange. The contaminated carbon has been stored with no designated final disposal.

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Coalite Chemicals--England. In 1968, dioxin-contaminated chemicals were released in an explosion in a trichlorophenol reactor inside the Coalite Chemicals plant in Derbyshire, England. Seventy-nine workers around the plant reportedly showed signs of chloracne during the next half year. Equipment and other materials found to be seriously contaminated were buried far down an abandoned coal mine shaft. In 1969, two workers developed chloracne during construction of a new building at Coalite. The source of their exposure was traced to a metal vessel that had been salvaged from the contaminated facility. The vessel reportedly had been extensively cleaned.

Phillips-Duphar--Amsterdam, Netherlands. In 1963, dioxin contaminated chemicals were released from a trichlorophenol reactor inside the Phillips-Duphar 2,4,5-TCP plant in Amsterdam. Twenty-eight employees were reported to have chloracne. Extensive measures were implemented to decontaminate and reconstruct contaminated equipment and areas in the building. However, animal toxicity testing showed that dioxin contamination was still present and the plant was demolished. The rubble was embedded in concrete and placed into three barges whose holds were hermetically sealed. The barges were towed out into the Atlantic ocean near the Azores and sunk in deep water.

Badische Anilin and Soda-Fabrik (BASF)--Ludwigshafen, Germany. In 1953, dioxin-contaminated chemicals were released from a trichlorophenol reactor inside the BASF plant in Ludwigshafen, Germany. This resulted in 75 cases of chloracne. Decontamination of the building was attempted. This was determined to be unfeasible and the building was demolished in 1986.

Dow Chemical--Midland, Michigan. In 1964, 49 out of 61 Dow workers exposed to dioxin-contaminated trichlorophenol developed chloracne. Dow is currently being investigated for

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dioxin contamination around its Midland, Michigan, chemical manufacturing facility.

Monsanto--Nitro, West Virginia. In 1949, dioxin-contaminated chemicals were released after a pressure buildup in a tri-chlorophenol reactor inside Monsanto's Nitro facility. One hundred twenty-two workers reportedly developed chloracne as a result of this contamination.

A study was released in May 1984 of 204 workers exposed to TCDD in cleanup operations at Nitro. The study concluded that, in the 35 years since their exposure, these workers had not developed more health problems than workers not exposed to TCDD.

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